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and
IDENTIFICATION SECTION

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The Plant Disease Reporter is issued as a service to plant pathologists throughout the United States. It contains reports, summaries, observations, and comments submitted voluntarily by qualified observers. These reports often are in the form of suggestions, queries, and opinions, frequently purely tentative, offered for consideration or discussion rather than as matters of established fact. In accepting and publishing this material the Plant Disease Epidemics and Identification Section serves merely as an informational clearing house. It does not assume responsibility for the subject matter.

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PLANT DISEASE EPIDEMICS AND IDENTIFICATION SECTION

Crops Research Division, Plant Industry Station, Beltsville, Maryland
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CAUSES OF CORN STALK ROT IN ILLINOIS

Benjamin Koehler[1] and G. H. Boewe[2]

Summary

In surveys for stalk rots in Illinois corn fields, Diplodia zeae was found to be the most prevalent pathogen and also the principal parasitic cause of premature death of plants. However, Gibberella zeae also was very important, being most prevalent two years in the eleven during which studies were made, and equal to D. zeae in three other years. Several other definitely parasitic causes of stalk rot were found but their average prevalence was very low.

The preponderance of stalk rot caused by Gibberella zeae (Schw.) Petch observed in 1946 (1) gave the writers a good opportunity to become well acquainted with this disease. In the absence of fruiting bodies of the causal fungus, there were no consistent definite external symptoms on the stalks by which to differentiate G. zeae infection from that caused by Diplodia zeae (Schw.) Lév. Sometimes G. zeae caused a target board effect as dark brown concentric half-rings extending up or down from a node, but this was the exception rather than the rule. When cut open, some but not all Gibberella-infected stalks were found to show a little pink color in a part of the rotted area. Since the two stalk rots are easily confused when the causal fungus is not fruiting, surveys were undertaken with the plan of making laboratory isolations from non-fruiting lesions on corn stalks.

Two different kinds of surveys were made, one in late summer when healthy plants were still fully green except for the ear husks in some cases, and a second made when the corn was mature.

The first survey reported here, made at the time the corn was mature (Table 1), is a continuation of similar surveys made by the Illinois State Natural History Survey for many years, except that since 1946 samples of non-fruiting stalk rot were brought to the laboratory. The pathogens were fruiting on many of the rot damaged stalks at the time of the late season surveys, and the cause of some of the non-fruiting stalk rot could also be determined at that time. Thus most of the identifications were made in the field.

Stalks were considered rot damaged when they showed definite rot discoloration on the exterior, or were soft near the base when pressed with the fingers, or showed numerous fungus fructifications. One hundred stalks were examined in various parts of one field in a county, and usually about half of the counties of the State, taken somewhat in checkerboard fashion, were visited. Stalks to be plated were cut off about an inch below the soil and the lowermost 18 inches brought to the laboratory. All leaves and roots were then removed, the plants were washed with a brush under running water and set to drain. They were then swabbed with $HgCl_2$, 1:1000, and usually three pieces were plated from the discolored areas. This was done by cutting away the surface with a flamed scalpel and transferring a fragment of tissue from beneath the surface to acidified potato dextrose agar. In some cases where it seemed advisable, stalks were split open and pieces plated from the interior. The plates were kept at room temperature for 5 days after which they were read, except for some fungi that required more time before identification could be made. Not all fungi could be identified.

The data in Table 1 show the percentage of stalks infected with each of a number of fungi. In some cases, especially in identifications made in the field, only one kind of infection was observed per stalk. Gibberella exceeded Diplodia only in 1946 and 1955. In three successive years, 1951, 1952, and 1953, the two were practically equal in prevalence. The 11-year average for the two was Diplodia zeae, 31.0 and Gibberella zeae, 25.8 percent of all stalks examined.

A limited number of fruiting specimens also were collected from various counties for microscopic verification at a later time. Of about 150 such specimens identified as Gibberella zeae in the field by the perithecia, all those that were mature proved to be this fungus. Up to the present, no perithecia of Gibberella fujikuroi (Saw.) Wr. have been identified in Illinois. Since the imperfect stage, Fusarium moniliforme Sheldon, was the stage of the fungus found

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Table 1. Corn stalk rot infection at maturity: fungi identified by fructifications, symptoms, or by plating; percentages based on random stalks examined, rotted or sound, in state-wide surveys.

Fungus / Year	Percent of stalks infected											
	1946	1947	1948	1949	1950	1951	1952	1953	1954	1955	1956	
Number stalks examined:	5700	4700	3400	4300	4800	3700	5100	5100	4700	4600	5100	Average
Diplodia zeae	17.8	33.2	39.7	52.5	42.7	34.6	19.4	20.1	34.6	18.9	27.3	31.0
Gibberella zeae	36.7	25.7	17.6	31.5	26.8	35.3	18.2	18.9	19.4	34.0	20.2	25.8
Sclerotium bataticola	0	.6	.4	.1	0	0	.5	1.8	.7	4.3	1.3	.9
Fusarium spp.[a]	5.0	9.6	5.2	2.9	3.7	4.9	3.7	2.4	3.7	3.8	3.1	4.4
Nigrospora oryzae	2.5	6.5	5.0	5.9	7.7	5.5	2.7	4.2	4.9	11.5	5.0	5.6
Helminthosporium spp.	(b)	(b)	.5	.6	(b)	.3	1.0	.2	1.0	.8	.4	.6
Phaeocytosporella zeae	.3	2.3	1.4	1.4	.9	.7	.7	1.1	.6	2.8	0.3	1.1

[a] Primarily F. moniliforme.
[b] No data available.

Table 2. Corn stalk rot infection in plants dying prematurely: fungi identified primarily by plating; percentages based on infected stalks only, collected in state-wide surveys.

Fungus / Year / Number stalks examined	Percent of stalks infected: 1948 (423)	1949 (723)	1950 (954)	1951 (1254)	1952 (1148)	Average
Stalks infected with only one fungus						
Diplodia zeae	23.7	29.2	11.2	7.9	17.2	17.8
Gibberella zeae	8.6	4.8	11.6	9.1	12.0	9.2
Sclerotium bataticola	1.7	0.1	0	0.3	2.1	0.8
Fusarium moniliforme	1.0	1.4	2.4	0.2	0.5	1.1
Nigrospora oryzae	0	.1	0	0	0	.02
Helminthosporium spp.	0	.1	0	.1	.1	.06
All fungi isolated, one or several per stalk						
Diplodia zeae	81.3	83.5	70.1	58.1	63.5	71.3
Gibberella zeae	24.4	23.1	47.5	58.5	38.2	38.3
Sclerotium bataticola	6.9	2.6	0	.9	5.7	3.2
Fusarium moniliforme	31.8	37.6	42.2	37.4	13.2	32.4
Nigrospora oryzae	6.4	11.8	16.0	33.2	12.2	15.9
Helminthosporium spp.	6.4	9.2	7.5	5.6	8.7	7.5
Phaeocytosporella zeae	1.6	1.6	.9	0.3	13.1	3.5
Trichoderma spp.	11.6	11.4	9.5	10.3	18.9	12.3
Others	10.6	1.5	4.9	.9	2.7	4.1
Unidentified[a]	5.8	5.5	4.8	15.8	11.2	8.6

[a] Some of these isolates were later identified as Pyrenochaeta terrestris (Hansen) Gorenz, Walker, & Larsen.

penetrating various parts of the corn plant in these studies, this name is used here as is proper under international rules of nomenclature (2).

In 1948 an additional type of survey was instituted for a 5-year period to determine, as far as possible, which fungi were responsible for stalk rot that resulted in premature death of plants. This survey was usually started at the beginning of the fourth week in August and continued for 4 weeks. In this survey, six stalks from prematurely dying plants in each of three fields in a county were collected. In the majority of cases the leaves were dying but the stalks were still green. Well separated fields were chosen but could not be taken entirely at random because: (a) fields had to show some plants dying prematurely from stalk rot, and (b) fields with leaves becoming dry from maturity, drought, leaf blight or other causes were not suitable. Satisfactory fields could be spotted fairly well from the highway. In better than nine-tenths of the fields where dying plants occurred scattered among fully green ones, the cause was stalk rot. In a few fields, however, such dying was due to other causes that could not readily be determined. Counties representative of the State, as mentioned before, were worked and all stalks on which this early season survey is based were brought to the laboratory. Rarely were any fungi fruiting on the stalks at that time.

The rots were well established in these plants that showed evidences of premature dying, and in the majority of cases more than one fungus was isolated from different lesions on the same stalk. Sometimes, moreover, two or even three fungi could be recognized as growing out of the same fragment plated. Therefore, with regard to causation, the upper half of Table 2 is of special interest since these are the stalks from which only one kind of fungus was isolated. The percentages are based on the stalks brought to the laboratory and have no relation to prevalence of prematurely dead plants in the fields. The latter would change from day to

day at that time of the year.

In one out of every four stalks from which Diplodia zeae was identified, it was the only pathogen present. Similar ratios were observed for Gibberella zeae, and for Sclerotium bataticola Taub. (Macrophomina phaseoli (Maubl.) Ashby). None of the other fungi isolated approached this ratio, indicating that the three mentioned were the principal pathogens. Although Fusarium moniliforme was associated primarily with other fungi, there nevertheless was an appreciable number of stalks with only that one infection, indicating that F. moniliforme by itself may cause some stalk rot.

Diplodia zeae and Gibberella zeae have caused serious stalk rot losses at times in certain fields all over the State. The ratio of Gibberella rot to Diplodia rot has tended to be a little higher toward the northern end of Illinois than farther south. Sclerotium bataticola also has occasionally been the principal cause of serious stalk rot damage in some Illinois corn fields, but primarily only in the southern half of the State and in years with hot summers. On the whole, its importance as compared to Diplodia and Gibberella (Tables 1 and 2) has been minor.

The prevalence of stalk rots varies not only from year to year (Table 1) but damage becomes noticeable earlier in some years than in others. With respect to Gibberella zeae, it has been noticed in particular that when infection comes early it can be very destructive, causing much lodging. At other times, there may be much G. zeae fruiting at maturity with comparatively little damage. In 1952 for instance, G. zeae was not nearly so prevalent as Diplodia zeae in the early survey (Table 2) but later the prevalence of the two became equal (Table 1).

The Helminthosporium found invading corn stalks appeared to be associated with senescence. Specimens sent to A. J. Ullstrup were identified as primarily H. carbonum Race II, Ullstrup. Phaeocytosporella zeae Stout (3) appeared to be weakly parasitic and of little economic importance.

There are other stalk rots in Illinois besides those mentioned, but they are usually of little importance. These are the hot weather rots occurring in midsummer, caused by Pythium butleri Subr. and Erwinia dissolvens (Rosen) Burkh. These two diseases are rather confusing as the symptoms appear to be the same. No critical study of the two has been made in Illinois, but both organisms have been isolated (E. dissolvens was identified by H. R. Rosen). No surveys have been made at the time these rots are active, and usually when found later, it was too late for making isolations.

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DEPARTMENT OF PLANT PATHOLOGY, UNIVERSITY OF ILLINOIS, AND THE ILLINOIS STATE NATURAL HISTORY SURVEY

CORN DISEASES AND THEIR IMPORTANCE IN MINNESOTA IN 1956[1]

J. E. DeVay, R. P. Covey, and P. N. Nair[2]

During recent years, the production of corn in Minnesota has greatly increased, and in terms of production value corn had an estimated value of 412 million dollars for a total production of 330 million bushels in 1956 or nearly half of the total value of all crops (1). Associated with this rapid increase in corn production have been changes in methods of growing corn. In the major corn growing areas of Minnesota, corn is commonly grown on the same fields in successive years with dense stands of about 18 to 22 thousand stalks per acre. To maintain the fertility of these fields, large amounts of fertilizers are used, occasionally with no regard to the balance of available mineral elements in a particular soil. As a result of these practices, a marked increase in the damage caused by various diseases and pests of corn has been apparent. For example, in the southern part of the State, growing corn after corn has resulted in increased root injury from corn rootworms; this in turn has resulted in severe root and basal stalk rot of corn in some fields, as the rootworms provide easy avenues of entrance into the roots for root and stalk rotting fungi. Stalk breakage and lodging increased sharply, depending on the corn hybrid grown, when stalk populations were increased from 18 to 24 thousand stalks per acre (2). The standing ability of various hybrids has also been decreased by using excessive amounts of nitrogen fertilizer in soils previously cropped to alfalfa which have a netural or slightly alkaline reaction. Preliminary experiments by the authors have indicated that the standing ability of corn stalks on fields such as these can be improved by decreasing the nitrogen to potash ratio.

The results of recent surveys on the losses caused by different corn diseases and the relative importance of pathogens involved in these diseases are discussed in the following paragraphs.

Seedling Blight -- The reduction in stand caused by seedling blight in 1956 was considerably less than that in 1955 when fields having 10-15 percent of blighted corn seedlings were common. In May and June of 1955, most corn growing areas in Minnesota were deficient in rainfall and the low soil moisture slowed the development of corn seedlings, increasing their susceptibility to root and crown rots. In contrast, in 1956, soil moisture was adequate for seedling growth and losses caused by seedling blight were negligible.

Root and Stalk Rots -- Root and stalk rots continue to be Minnesota's most important corn diseases. The severity and prevalence of these diseases, often indicated by broken and lodged stalks, was as great in 1956 as it was in 1955. Fields having as high as 90 percent broken stalks were observed, particularly in the southwestern part of the State. Actual losses in yield are difficult to determine because root and stalk rots can cause a reduction in yield of standing plants up to 100 percent without conspicuous disease symptoms. Moreover, a sizeable reduction in yield usually results due to the dropping of ears from rotted shanks and the loss of ears from broken stalks that are missed by pickers. In some instances this loss is minimized by pasturing livestock in fields where considerable stalk breakage has occurred. However, when ears are in contact with moist soil, molds develop rapidly and rot the ears, frequently making them unfit for animal feed. The extent of stalk breakage in fields is often reflected the next year by volunteer corn in following crops such as soybeans. During the past 2 or 3 years, it has been common to see fields of soybeans which were almost hidden by volunteer corn; considerable labor is necessary to get soybean fields such as these ready for combining. When volunteer corn is not cut before harvesting soybeans, efficient operation of the combine machinery is affected and mixtures as high as 50 percent corn and 50 percent soybeans have been reported. Thus, when assessing the damage and loss caused by stalk and root rots of corn, it is important to consider not only direct losses such as the reduction in yield but also losses in time and labor and quality and yield of following crops. A conservative estimate of losses caused by these diseases in Minnesota in 1956 is 10 percent of the value of the corn crop.

Surveys during the 1955 and 1956 growing seasons were made of the relative importance

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Table 1. Organisms isolated from samples of rotted stalks and roots of corn plants collected from 109 locations in the major corn growing areas of Minnesota during 1955 and 1956.

Organisms isolated[a]	Percentage of samples from which isolates were obtained	
	1955[b]	1956[c]
Alternaria sp.	30	13
Aspergillus sp.		0.4
Bacteria	2	35
Botrytis sp.		0.4
Cephalosporium sp.		6
Chaetomium sp.	2	0.4
Cladosporium sp.		0.4
Diplodia zeae	18	3
Fusarium spp.[d]	98	80
Helminthosporium sp.	6	1
Mucor sp.	2	0.4
Nigrospora sp.		1
Penicillium sp.	8	1
Rhizoctonia sp.		0.4
Rhizopus sp.		1
Spicaria sp.		0.4
Trichoderma lignorum	14	

[a] Eight fungi from the isolations made in 1955 and 5 fungi from those made in 1956 were non-sporulating and are not identified.
[b] Isolations were made from 50 plant samples collected at 50 different locations and plated on acidified potato dextrose agar.
[c] Isolations were made from 269 samples collected at 59 different locations and plated on corn meal-peptone agar.
[d] In further culture tests involving the use of special agars and mixed cultures, perithecia of Gibberella zeae and Gibberella fujikuroi were found. These tests indicated that the predominant Fusarium sp. was F. graminearum.

of various fungal pathogens causing stalk and root rots of corn in Minnesota. Over 300 samples of diseased roots and stalks from 109 different corn fields located throughout the important corn growing areas of Minnesota were collected. Sections of tissue isolated from these samples were surface treated with a 1% solution of sodium hypochlorite for 1 to 2 minutes, then rinsed in sterile water and plated on corn meal agar or acidified potato-dextrose agar. Frequency of isolation of a given organism was based on the number of samples from which this organism was isolated. Fungi whose identity was not readily apparent were transferred to agar slants and later identified.

The results of surveys such as these (Table 1) do not give an accurate picture of the relative importance of organisms causing the initial infections, nor do they give an indication of the capacities of different fungi for parasitizing living tissues under natural conditions. However, these surveys give some indication of the kinds of fungi which grow as saprophytes in dead stalks prior to corn picking time. These fungi are important from the standpoint that they can weaken the dead stalks and cause considerable breakage before picking, particularly in dry years.

During the past 3 growing seasons, additional isolations were made from the marginal areas of rotted tissues in living plants. More than 200 stalk samples, collected from fields throughout the major corn growing areas of Minnesota, were tested. In all but three or four instances, Fusarium spp. were the only fungi isolated; in the other cases, we were testing our techniques and recovered Diplodia zeae along with Gibberella zeae from plants inoculated with mixed cultures of these fungi. These studies indicated that Gibberella spp. and Fusarium spp. are the most important causes of stalk rot in living plants in all stages of post-emergence development in Minnesota. Although certain isolates of Diplodia zeae as well as other fungi

have been highly virulent in controlled pathogenicity tests (3) and are common in corn fields, these organisms are relatively unimportant as compared with Gibberella spp. and Fusarium spp.

Smut -- Smut caused by Ustilago maydis continues to be an important corn disease problem in Minnesota, although in recent years losses have been reduced by increased resistance in commercial hybrids. Reduction in yield caused by smut was about 1 to 2 percent in 1956.

Leaf Blights and Rust -- Various fungus and bacterial leaf blights and rust were prevalent in all corn fields surveyed for corn diseases; however, these leaf diseases were not severe and losses were probably negligible in 1956.

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A DIAGNOSTIC KEY FOR THE IDENTIFICATION OF PHYSIOLOGIC RACES OF PUCCINIA RUBIGO-VERA TRITICI GROUPED ACCORDING TO A UNIFIED NUMERATION SCHEME

Rita Basile[1]

Johnston (1) recently proposed the consolidation of closely related physiologic races of Puccinia rubigo-vera (DC.) Wint.[2] f.sp. tritici (Erikss.) Carl. (= P. triticina Erikss.) into unified groups. Under this scheme, each of the unified groups would embrace those physiologic races which produce identical or similar infection types on five of the standard differential varieties. These critical varieties are: Malakof (C.I. 4898), Webster (C.I. 3780), Loros (C.I. 3779), Mediterranean (C.I. 3332), and Democrat (C.I. 3384).

A diagnostic key has been prepared to facilitate the identification of the unified groups on the basis of the above five varieties. The key, which follows, includes the 163 races described by Johnston and Levine (2) as well as 18 new races identified for the first time in Italy. The key was constructed on a dichotomous basis, providing for the differentiation of the varietal reactions into the following two classes: R, indicating resistance as expressed by the infection types 0, 1, 2; and S, indicating susceptibility as expressed by the infection types X, 3, 4. This is in accord with the scheme already used by Levine, Ausemus, and Stakman (3) for leaf rust of wheat; and by Levine and Cherewick (4) for leaf rust of barley.

Key -- Diagnostic key for the identification of unified groups encompassing physiologic races of Puccinia rubigo-vera tritici.

	Unified group Numbers
Malakof resistant	
Webster resistant	
Loros resistant	
Mediterranean resistant	
Democrat resistant	1
Democrat susceptible	23
Mediterranean susceptible	
Democrat resistant	20
Democrat susceptible	2
Loros susceptible	
Mediterranean resistant	
Democrat resistant	10
Democrat susceptible	16
Mediterranean susceptible	
Democrat resistant	4
Democrat susceptible	3
Webster susceptible	
Loros resistant	
Mediterranean resistant	
Democrat resistant	22
Democrat susceptible	26
Mediterranean susceptible	25
Loros susceptible	
Mediterranean resistant	
Democrat resistant	12
Democrat susceptible	27
Mediterranean susceptible	
Democrat resistant	19
Democrat susceptible	17

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[2] [Properly designated P. recondita Rob. ex Desm.]

Key -- (Continued).

Malakof susceptible
Webster resistant
Loros resistant
Mediterranean resistant
Democrat resistant 11
Democrat susceptible 21
Mediterranean susceptible 5
Loros susceptible
Mediterranean resistant 14
Mediterranean susceptible
Democrat resistant 24
Democrat susceptible 6
Webster susceptible
Loros resistant
Mediterranean resistant 18
Mediterranean susceptible 7
Loros susceptible
Mediterranean resistant
Democrat resistant 9
Democrat susceptible 15
Mediterranean susceptible
Democrat resistant 8
Democrat susceptible 13

The reactions of the five differential varieties to each of the unified groups are given in Table 1. The right hand column of this table lists the physiologic races comprising each of the unified groups.

Table 1. Reaction of the five varieties of wheat used to differentiate the unified groups of physiologic races of Puccinia rubigo-vera tritici.

Unified group number	Reactions of 5 differential varieties[a]					Internationally registered physiologic races and newly identified Italian races incorporated in the unified group numbers listed in the first column.
	Malakof	Webster	Loros	Mediterranean	Democrat	
1	R	R	R	R	R	1, 16, 53, 63, 120, 123.
2	R	R	R	S	S	2, 15, 25, 34, 59, 62, 127, 151, 153.
3	R	R	S	S	S	3, 12, 32, 44, 58, 61, 78, 84, 98, 102, 111, 141, 161.
4	R	R	R	S	R	4, 156, 163.
5	S	R	R	S	S	5, 52, 100.
6	S	R	S	S	S	6, 28, 39, 40, 83, 103, 105, 126, 144.
7	S	S	R	S	S	7, 60.
8	S	S	S	S	R	8, 48, 172(R26)[b]

Table 1. (Continued).

Unified group number	Reactions of 5 differential varieties[a]					Internationally registered physiologic races and newly identified Italian races incorporated in the unified group numbers listed in the first column.
	Malakof	Webster	Loros	Mediterranean	Democrat	
9	S	S	S	R	R	9, 10, 13, 19, 20, 23, 29, 31, 70, 108, 146, 147, 148.
10	R	R	S	R	R	11, 14, 26, 38, 74, 95, 131, 132, 133, 154, 155.
11	S	R	R	R	R	17, 36, 93.
12	R	S	S	R	R	18, 66, 68, 72, 96, 97, 106, 107, 134, 135, 138, 139, 164(R18), 166(R20), 168(R22).
13	S	S	S	S	S	21, 30, 35, 42, 54, 65, 77, 80, 82, 89, 94, 101, 104, 112, 113, 114, 115, 116, 122, 130, 145, 149, 150.
14	S	R	S	R	R	22, 37, 43, 49, 50, 64, 69, 110, 128.
15	S	S	S	R	S	24, 27, 41, 99, 117, 152.
16	R	R	S	R	S	33, 46, 51, 79, 158, 178(R32).
17	R	S	S	S	S	45, 57, 67, 76, 81, 85, 87, 88, 90, 136, 140, 142, 143, 162, 167(R21), 176(R30).
18	S	S	R	R	R	47, 91, 119, 121, 125.
19	R	S	S	S	R	55, 71, 73, 75, 86, 137, 157.
20	R	R	R	S	R	56, 92, 165(R19).
21	S	R	R	R	S	109, 160.
22	R	S	R	R	R	118, 174(R28), 177(R31).
23	R	R	R	R	S	124, 169(R23), 179(R33).
24	S	R	S	S	R	129, 180(R34).
25	R	S	R	S	S	159.
26	R	S	R	R	S	170(R24), 171(R25), 173(R27).
27	R	S	S	R	S	175(R29), 181(R35).

[a] Malakof (C. I. 4898), Webster (C. I. 3780), Loros (C. I. 3779), Mediterranean (C. I. 3332), Democrat (C. I. 3384).

[b] Grateful acknowledgment is made to Prof. C. O. Johnston for his letter of February 14, 1957 in which he furnished the international numbers from 164 to 181 for the new Italian races which had been provisionally designated as R18 to R35, inclusive.

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STATION OF PLANT PATHOLOGY, MINISTRY OF AGRICULTURE AND FORESTRY, ROME, ITALY

REPORT ON A STUDY OF THE HEALTH HAZARD ASSOCIATED WITH THE TREATMENT OF SEED GRAIN WITH ORGANIC MERCURY COMPOUNDS APRIL 10-27, 1956

G. J. Raschka[1]

The treatment of seed grain for the control of fungi has become an established practice in the farming community. Some of the most effective seed disinfectants contain an organic mercury compound as the active ingredient.. All of the treating encountered during this study was performed with liquid disinfectants as contrasted with the dry disinfectants in use at the time of a similar survey made in 1947.

Mercury and its compounds, either in the form of dust or vapor, are extremely toxic to humans. They may enter the body by ingestion with food, by inhalation of the dust or vapor or by prolonged contact with the skin. Fine dust from the grain itself may carry the mercury compound into the working atmosphere. Control of each of these means of entry into the body must become part of the treating procedure to prevent impairment of the health of exposed workers. The American Conference of Governmental Industrial Hygienists has adopted 0.1 milligram of mercury, as metallic or inorganic compounds, or 0.01 milligram of mercury, as organic compounds, per cubic meter of air as the limits that should not be exceeded for exposures of eight hours per day, day after day. The lower limit for organic mercury compounds was recently adopted and points out the greater potential hazard associated with the use of mercury compounds of the type used in seed treating. Absorption through the skin is also a greater problem with these organic compounds.

In 1947, 54 samples of air-borne dust and vapor were collected in 12 establishments treating seed grain with dry disinfectants. The results showed that the atmospheric mercury concentration varied from 0.05 to 4.7 with an average of 0.74 milligrams per cubic meter of air. The workers complained of early symptoms of mercury absorption (high protein foods such as eggs are distasteful, cigarette smoking is unpleasant, eye and nose irritation, some nausea), and it is assumed that more serious signs of chronic mercury poisoning were not apparent only because the seed treating season was short and the daily exposure was considerably less than eight hours per day.

In the present study, visits were made to 48 establishments that were believed to be treating grain. In 16 of those that were treating at the time of the visit, 42 air samples were collected. In one establishment, three samples showed atmospheric mercury concentrations of 0.53, 0.24 and 0.19 milligrams per cubic meter of air. In the remaining 39 samples in 15 places of business, the air-borne mercury concentrations varied from less than 0.01 to 0.08 with an average of 0.03 milligrams per cubic meter of air. The one group of high samples was separated out because they were not typical of the group. A logical explanation for these elevated results was not apparent from a visual observation of the treating operation. It is possible that gross contamination of the premises had occurred or that storage of treated grain had been the source of mercury vapor evolution.

A comparison of the results of the 1947 and the 1956 studies reveals a substantial reduction in the atmospheric mercury concentrations associated with the seed treating operation. This can be attributed to a considerable extent to the change in method from dry to liquid treating. An evaluation of the average value of 0.03 milligrams of mercury per cubic meter of air on a time-concentration basis suggests that the exposure compares favorably with the adopted limit of 0.01 milligram per cubic meter of air since in most establishments the actual treating time probably does not exceed one-third of the working day. At this level of exposure, attention must be given to the importance of ingestion with food and absorption through the skin as added modes of entry into the body. That total exposure is at the border line of chronic illness is substantiated by the acknowledgment of early symptoms by many of the workers exposed.

It is obvious from the above description of findings that precautionary measures are still necessary to avoid excessive exposure to the organic mercury treating materials. The following control measures are suggested:

1. The workers should be provided with and wear adequate respiratory protection for those operations involving intimate exposure to the disinfectant such as:

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maintenance on treater equipment, clean up of contaminated areas, and sacking of treated grain or other dusty loading or unloading operations. A chemical-cartridge type respirator containing a chemical capable of absorbing mercury vapor may be used.

2. The workers should wear protective clothing to prevent contact of the skin with the disinfectant. Rubber rather than cloth or leather gloves should be worn. Work clothes that may be contaminated should not be worn home but should be changed at the end of each day. Adequate facilities should be provided for washing the hands with soap and water immediately after performing each job where skin contact is expected or occurs.
3. Eating and smoking should not be done on the premises, or at least not in those areas where contamination can occur. Before eating, the hands should be washed with soap and water.
4. The treatment of seed grain should be carried out in areas that are well ventilated. In some cases, mechanical exhaust ventilation must supplement natural ventilation. Local exhaust ventilation may be required for some operations such as sacking. The loading of trucks outside of the building appears to be a practical way of reducing air-borne contamination in the working area.
5. A program of good housekeeping should be applied to all operations associated with the seed treating process to prevent the dispersion of mercury-laden dust or vapors into the working atmosphere and to reduce the possibility of ingestion or absorption of the material.

CONCLUSIONS

The results of this study indicate that a substantial reduction in air-borne mercury concentration occurred after the substitution of liquid for dry dust seed treating was made. The degree of contamination is still at the border line where early signs of mercury poisoning may occur among the workers. The foregoing suggestions are offered as a check list for further control of the potential hazard involved.

MINNESOTA STATE DEPARTMENT OF HEALTH, DIVISION OF ENVIRONMENTAL SANITATION, UNIVERSITY CAMPUS, MINNEAPOLIS 14

INCIDENCE OF BARLEY STRIPE MOSAIC IN MONTANA FOR TWO YEARS AS INDICATED BY SEED ASSAYS[1]

H. S. MacWithey, Jr., I. K. Mills, and M. M. Afanasiev

Barley stripe mosaic, a seed-borne, mechanically transmissible virus disease, was identified in 1950 (4). Severe reductions of yield have been shown to occur in barley or wheat infected with this disease (2, 3). A barley seed-testing program for stripe mosaic in Montana was initiated in 1954 as a result of the recognition that severe yield losses could result from this disease and that the potential for spread of the disease was quite high. A previous paper (1) described the results of seed assays conducted in 1954-55 to determine the incidence of stripe mosaic in barley seed produced in Montana. This testing program was continued during the winter and spring of 1955-56 on the 1955 crop using the same assay procedure as described by Afanasiev (1).

The predominant barley variety assayed in these tests was Compana which, on the basis of acreage planted, is the most important barley variety grown in Montana. Barley varieties other than Compana tested the first year were Titan, Gem, Vantage, Moore, and Moravian. During the second year, Titan, Moravian, Vantage, Sultana, Spartan, Moore, Glacier, Wolf, and Husky varieties in addition to Compana were assayed. One Titan sample from the 1954 crop was infected with stripe mosaic, and of the 1955 crop, 3 samples of Titan, 1 of Moravian, 1 of Sultana, and 1 of Glacier carried virus. In no case was the level of infection of these samples, expressed as the percentage of seed infected, found to be higher than 5 percent.

For the first year of the tests, 219 samples of Compana variety barley from the 1954 crop were tested and 120 (54.8 percent) were found to be infected with stripe mosaic while 99 (45.2 percent) were apparently disease-free. The next year, out of 482 samples of Compana from the 1955 crop, 246 (51.0 percent) contained virus while 236 (49.0 percent) were without detectable stripe mosaic. The distribution of the diseased Compana samples into ranges based upon levels of infection of the seed with stripe mosaic for 1954 and 1955 is given in Table 1.

Table 1. Levels of infection of Compana seed lots with stripe mosaic, 1954 and 1955.

Level of infection	Number of samples	
	1954	1955
0-5%	59	182
5-10%	25	34
10-25%	29	22
25-50%	7	8
50-100%	0	0
Totals	120	246

Included in the 1955-56 tests were 101 samples produced from seed assayed and found to be disease-free by the tests conducted in 1954-55. No virus was detected in 65 (64.4 percent) of these samples but 36 (35.6 percent) showed levels of infection ranging from 1 to 5 percent with the exception of 1 sample which showed 7 percent of the seed infected with stripe mosaic. Seed produced from 9 Compana seed lots found to be diseased in 1954-55 were also assayed in 1955-56. No appreciable change in the levels of infection was observed for 8 samples in the 1 to 5 percent infection range, but one sample showed an increase of diseased seed from 9 percent in the 1954 crop to 14 percent in the 1955 crop.

A comparison of the percentages of diseased Compana samples falling into various ranges based on levels of infection for 2 years, Figure 1, shows an increase in the proportion of light infections (i.e., 1-5 percent) in the 1955 crop, with a concomitant reduction in the heavier in-

[1] Contribution from Montana State College, Agricultural Experiment Station, Bozeman, Montana, Paper No. 404 Journal Series.

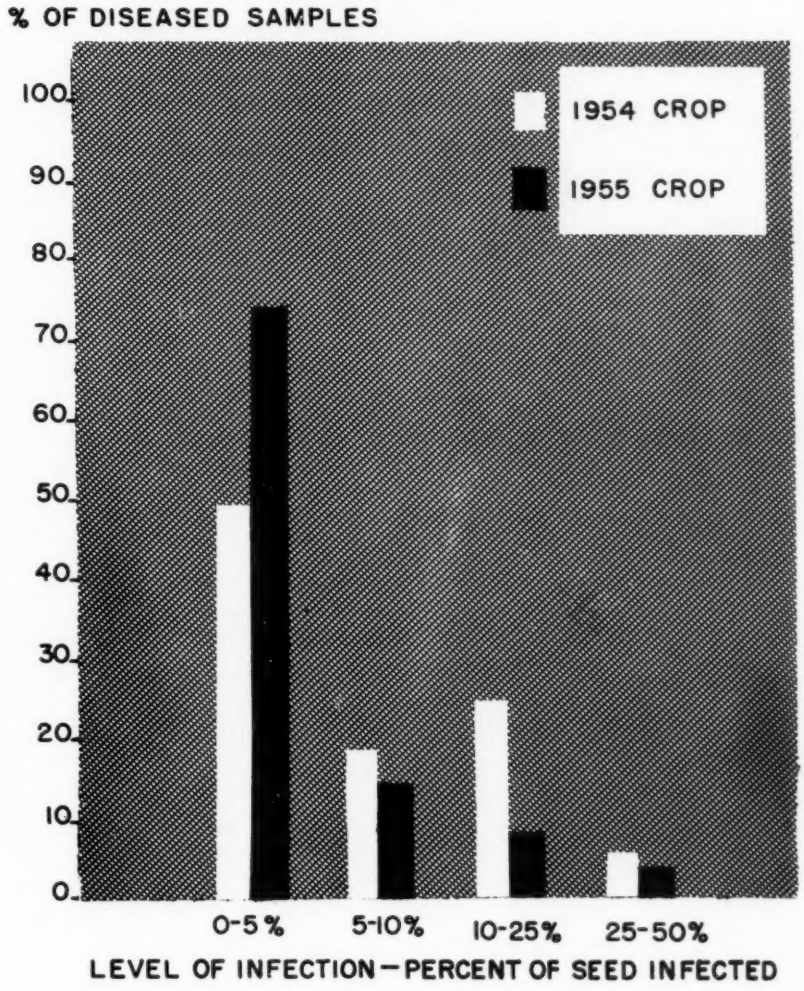

FIGURE 1. Levels of infection of Compana seed lots with stripe mosaic, 1954 and 1955.

fections. These data, together with the observed reduction in the percentage of the 1955 samples carrying virus, indicate that it may not be possible to completely eliminate stripe mosaic by planting seed which was found to be virus-free. However, the potential for spread and increase of the virus may be reduced by detection and elimination of the heavily infected seed lots.

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MONTANA STATE COLLEGE, AGRICULTURAL EXPERIMENT STATION, BOZEMAN, MONTANA

THE SPREAD OF LAMBERT MOTTLE VIROSIS IN LAMBERT AND PEERLESS VARIETIES OF SWEET CHERRIES IN MONTANA[1]

M. M. Afanasiev and I. K. Mills

Lambert mottle, a serious virus disease of Lambert cherries, was described in Canada in 1945 (3). The first observation in Montana of symptoms resembling those of this disease occurred in 1946 in one orchard on the south shore of Flathead Lake near Polson. By 1950 similar symptoms were found on trees in several other sweet cherry orchards in the Flathead Lake region (1). The virus nature of the disease and the similarity to Lambert mottle were proven by budding and transmission experiments. Symptoms of Lambert mottle virosis have been observed only on the Lambert and Peerless varieties of cherries in Montana, but other varieties may be latent carriers.

Detailed surveys of plots in certain sweet cherry orchards have been carried out for several years, and during this time variations of the severity of symptom expression on individual trees and the spread of Lambert mottle in orchard plots have been recorded. The rates of increase of Lambert mottle within three orchards for the years of the surveys are shown in Table 1. These data show that new infections, as indicated by symptom expression by individual trees, continually occur, and the percentage of the total trees infected constantly rises. The rate of spread, expressed as the percentage of the original trees becoming newly infected, varies in different years from about a low of 1 percent to a high of almost 21 percent.

Table 1. Increase of Lambert mottle virosis in Lambert and Peerless cherry trees in survey plots of three orchards.

Year	Number of trees		Newly infected trees		Total infection of original trees percent
	Healthy	Diseased	Number	Percent of original trees	
Orchard C					
1946	101	7	7	6.5	6.5
1947	101	7	0	0.0	6.5
1948	101	7	0	0.0	6.5
1949	99	9	2	1.9	8.3
1950	81	27	18	16.7	25.0
1951	80	28	1	0.9	25.9
1952	77	31	3	2.8	28.7
1953	70	38	7	6.5	35.2
1954	66	42	4	3.7	38.9
1955	63	45	3	2.8	41.7
1956	57	51	6	5.6	47.2
Orchard H					
1950	156	19	19	10.9	10.9
1951	152	23	4	2.3	13.1
1952	150	25	2	1.1	14.3
1953	137	38	13	7.4	21.7
1954	132	43	5	2.9	24.6
1955	113	62	19	10.9	35.4
1956	108	67	5	2.9	38.3
Orchard R					
1954	98	23	23	19.0	19.0
1955	93	28	5	4.1	23.1
1956	68	53	25	20.6	43.8

[1] Contribution from Montana State College, Agricultural Experiment Station, Bozeman, Montana. Paper No. 405 Journal Series.

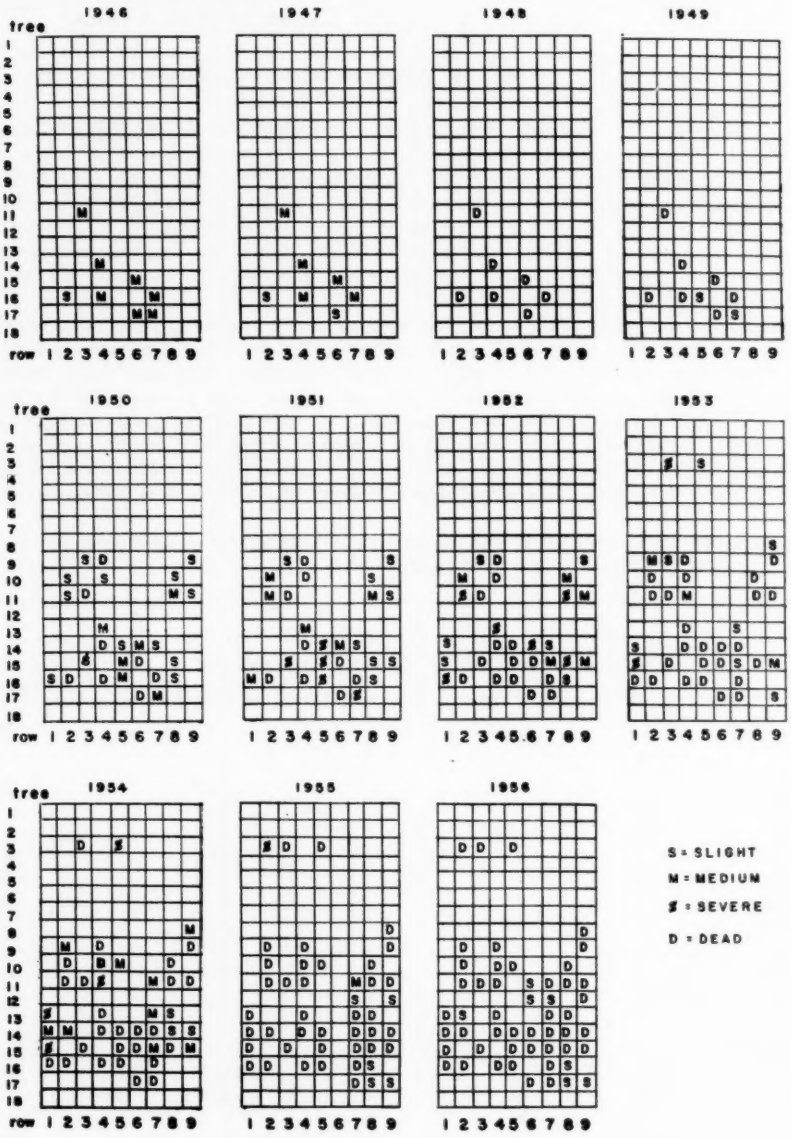

FIGURE 1. Spread of Lambert mottle on Lambert cherry trees in orchard "C", Lake County, Montana.

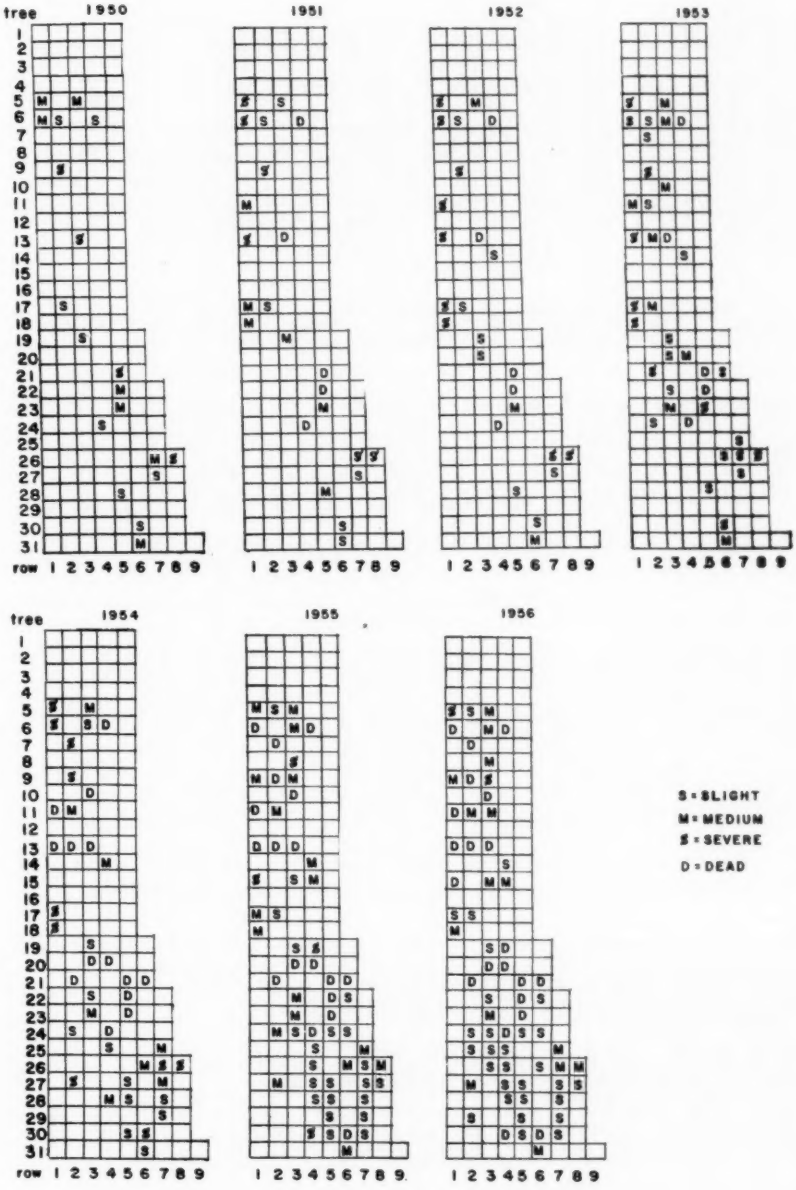

FIGURE 2. Spread of Lambert mottle on Lambert and Peerless cherry trees in orchard "H", Lake County, Montana.

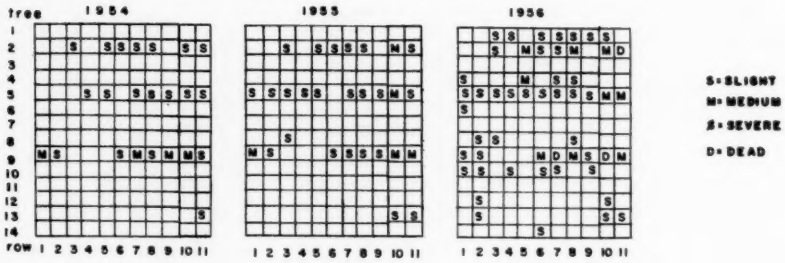

FIGURE 3. Spread of Lambert mottle on Lambert cherry trees in orchard "R", Lake County, Montana.

Afanasiev and Morris (2) in 1954 reported that the direction of spread of this disease within orchards in general follows a radial pattern around an initial site of infection. The spread and symptom severity of Lambert mottle virosis in Lambert and Peerless trees in survey plots of the three different orchards are shown in Figure 1, 2, and 3. In these survey plots some trees infected with Lambert mottle died and were replaced during the period of the surveys.

In Orchard C (Figure 1), the first orchard in which Lambert mottle was observed in Montana, it is noted that the spread of the virosis tends to be radial about certain more or less well-defined centers of infection, but with occasional outbreaks some distance away from these points. Orchard H (Figure 2) showed a greater diffusion of the disease when it was first found in this orchard in 1950. Here, too, Lambert mottle appeared to spread radially from the initially observed infected trees. A somewhat different pattern of dissemination apparently occurred in Orchard R (Figure 3). Although this orchard had been surveyed for several years previously, Lambert mottle was not found until 1954. At this time the disease in the survey plot was restricted mainly to three rows, but the majority of the trees showed symptoms. The increase in the number of trees expressing Lambert mottle symptoms was slight the following year and again tended to follow the pattern of the rows. However, in 1956 a greater spread was observed, and the pattern was similar to those observed in the other two orchards.

The specific vector or vectors which disseminate Lambert mottle virus are unknown, but the evidence seems to indicate that a factor of relatively limited range is involved.

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MONTANA STATE COLLEGE, AGRICULTURAL EXPERIMENT STATION, BOZEMAN, MONTANA

ASTER YELLOWS VIRUS AFFECTING STRAWBERRIES IN ARKANSAS[1]

J. P. Fulton

Abstract

The occurrence of aster yellows virus in strawberry plantings in Arkansas since 1953 indicates that this disease is commonly present although rarely in damaging amounts. "August black root" or "spot dying" was also demonstrated to be caused by the aster yellows virus. The aster yellows virus was verified by dodder transmission from strawberry to periwinkle and back to strawberry.

INTRODUCTION

A strawberry disease whose symptoms resemble those recorded for the aster yellows virus was first reported in Arkansas by Smith (8). The present report concerns the field development of this disease since 1953, verifies the cause to be the aster yellows virus, and describes additional symptoms.

Frazier and Thomas (2) first reported the occurrence of aster yellows virus in strawberry plantings in California. Plakidas in 1951 (5) had previously noted "chlorotic phyllody" from Louisiana, the symptoms of which are identical with those caused by the aster yellows virus. In 1953 Posnette (6) reported the occurrence of "green petal" in England and Scotland. Frazier and Posnette (3) showed that green petal was caused by a yellows virus similar to, but distinct from, the strains of aster yellows virus ordinarily encountered. In 1955 Gourley (4) observed green petal in Nova Scotia.

FIELD OCCURRENCE IN ARKANSAS

The occurrence of aster yellows virus in strawberries in 1953 and 1954 was reported by Smith (8). Attention was focused on the problem in 1954 because of the widespread nature of the outbreak. During that spring the disease was present in all strawberry plantings observed. In a few cases as many as 20 percent of the plants were affected. Detailed observations on several strawberry fields indicated that the virus did not spread from diseased to healthy plants in the field. The disease was self-limiting in that all infected plants eventually died. In 1955 and 1956 aster yellows virus was noted in only a few fields, affecting a relatively few plants. It would appear that the disease has returned to the status of an oddity as far as the spring development of symptoms is concerned.

TRANSMISSION

Aster yellows virus was readily transmitted from diseased to healthy plants by stolon grafts, stolon to petiole grafts, and by excised leaflet grafts of the type described by Bringhurst and Voth (1). The only difficulty encountered was that diseased plants died so rapidly that sufficient time for a graft union was often lacking. The incubation period following a successful graft varied from 36 to 80 days.

Symptoms were first noted as yellowing, cupping and stunting of new leaves. If the plant survived the initial shock, it persisted with dwarfed leaves and a multiple crown. The abnormal flower symptoms, so characteristic of the disease in the field, were rarely observed in the greenhouse following graft transmissions. Symptoms in the commonly used strawberry virus indicator Fragaria vesca L. (East Malling Clone) were essentially the same in that the plant was reduced to a multiple crown covered with dwarfed leaves. The symptoms were not readily distinguishable from those produced by the strawberry virus complex of mottle and epinasty.

Transmission was accomplished by dodder (Cuscuta campestris Yuncker) from strawberry to periwinkle (Vinca rosea L.) and back to strawberry. Leaf hopper transmission was not attempted.

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[1] Published with the approval of the Director of the Agricultural Experiment Station.

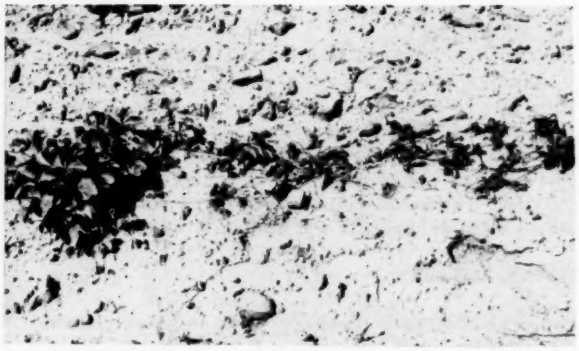

FIGURE 1. "Spot dying" photographed in August, 1956. The dead or dying plants in the center of the picture are all connected by runners. Healthy plants are evident to the left and extreme right in the row.

FIGURE 2. Strawberry plant showing first evidence of August black root. Leaves are cupped and roots are dead.

MIDSUMMER FIELD SYMPTOMS

During 1954 and 1955 all strawberry plants affected by aster yellows virus in the field had died by July and there was apparently no further evidence of the virus the remainder of the year. Evidence obtained in 1956, however, indicated that this virus may commonly be responsible for the death of plants in many fields during July and August. This disease has been called locally "August black root" or "spot dying". The problem of spot dying has been studied for many years. Diseased plants are most commonly noted during late July or August. This disease is characterized by a sudden wilting and death of the affected plants. All plants connected to an infected plant by runners die, thus forming a spot of dead plants (Fig. 1). Dead plants are often intermingled with healthy plants, derived from a different mother plant. When dying plants are first noted the roots are black and dead (Fig. 2). No parasitic fungi or nematodes have been found to be consistently associated with the dying plants.

During August of 1956 "spot dying" was noted in a number of fields. In one case collections were made of plants in the first stages of wilting. From these, by means of leaf and runner grafts, the disease was reproduced in healthy strawberry plants. Under greenhouse

conditions symptoms were essentially the same as those of strawberry plants affected by the aster yellows virus from spring collections. The presence of aster yellows virus was confirmed by means of dodder transmission to periwinkle.

It is not known how much of the "spot dying", often encountered in strawberry fields in midsummer, can be attributed to the aster yellows virus. It is the opinion of the author that aster yellows virus is responsible for the major amount of "spot dying" for which there is no other obvious explanation.

OTHER DISEASES RESEMBLING ASTER YELLOWS IN STRAWBERRY

In the course of studying aster yellows in strawberries it was noted that several strawberry diseases might easily be confused. Frazier and Thomas (2) pointed out that the foliage symptoms of the aphid transmitted strawberry viruses, in varieties used in California, resemble foliage symptoms produced by the aster yellows virus. It was believed that similar confusion did not exist in Arkansas since Blakemore and most of the other commonly grown varieties developed no obvious symptoms when infected with the aphid transmitted viruses. Aster yellows produces severe symptoms in these varieties as noted above. Another virus resembling the foliage symptoms of aster yellows was encountered, however, which did produce foliage symptoms in Blakemore and several other varieties. It differed from aster yellows in having a shorter incubation period (4 weeks), producing very mild symptoms or no symptoms at all in the indicator Fragaria vesca, East Malling Clone, and in failing to produce any degree of phyllody of flowers. This virus has not been identified.

It was pointed out by Slack, Seymour, Fields and Fulton (7) that summer dwarf, caused by the foliar nematode Aphelenchoides besseyi Christie, may have symptoms resembling those produced by the aster yellows virus. The symptoms of summer dwarf are most evident in the fall whereas aster yellows virus in strawberry has been noted most commonly in the spring and summer.

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TOBACCO RINGSPOT VIRUS FROM IRIS AND EASTER LILY

R. V. Travis[1] and Philip Brierley[1]

In June 1956 a ringspot virus was recovered from Iris germanica L., variety Great Lakes from New Jersey, by sap inoculations to bean (Phaseolus vulgaris L.). This was briefly described as iris ringspot virus[2]; mutual antagonism tests since showed that this is a strain of tobacco ringspot virus (TRspV). No symptoms were detected in the naturally infected iris plants. Experimental attempts to infect I. germanica hybrid seedlings by sap transfer from infected tobacco failed.

The symptoms in various test plants are similar to those produced by the type strain ATCC No. 98 of TRspV but differ in severity. In bean, tobacco, Gomphrena, and snapdragon the symptoms of the iris strain are milder than those of the type virus, but in Chenopodium they are more severe and include systemic leaf distortion. The physical properties of the iris strain differ slightly from those of the type strain of TRspV.

In January 1957, 25 plants of Georgia Easter lilies (Lilium longiflorum Thunb.), grown in Georgia, were indexed by sap inoculation to tobacco, primarily to determine whether they were free from cucumber mosaic virus (CMV). No CMV was detected, but a few broken gray-ring local lesions appeared in tobacco inoculated with sap from one lily. Subinoculation from these local lesions to young tobacco produced systemic gray rings followed by brown rings and recovery. The virus recovered from the same lily plant in February again produced local lesions only in tobacco. From systemically invaded tobacco it was sap-transferred to Creole Easter lily and reisolated from this lily in tobacco. No symptoms appeared in the naturally infected Georgia lily or in the experimentally infected Creole lily during a full season of growth in a greenhouse.

The ringspot strain from lily is less virulent on test plants such as tobacco, bean, snapdragon, and Chenopodium than the ringspot strain from iris or the TRspV type; the symptoms produced are of the same general type but considerably milder. In Gomphrena the lily strain is more virulent than the iris strain, but less so than the type virus. All three strains produce systemic symptoms in Gomphrena. In Chenopodium the lily strain forms local lesions only; the other two invade this plant systemically. Tobacco that recovered from the lily virus symptoms after infection of 30 days or longer was protected against superinfection by the iris ringspot or by TRspV.

Simultaneous-infection tests[3] were made in beans and tobacco to see whether additive effects could be detected with mixtures of the iris and lily strains with each other or with the type TRspV. No increase in severity of symptoms was recognizable in any combination of strains, the more virulent of the paired strains determining intensity of symptoms in each instance. Failure to produce additive effects in combination thus supports the evidence from cross-protection tests that the three are strains of TRspV.

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CITRUS-ROOT NEMATODE A NATIVE TO FLORIDA SOILS

B. G. Chitwood and W. Birchfield

Tylenchulus semipenetrans was first discovered in California on citrus roots by J. R. Hodges, Horticultural Inspector for Covina county (7). Cobb (2) gave attention to the anatomy, life history and distribution of this species as determined by root samples from various citrus-growing regions, and showed that it has been found in all of the major citrus-producing areas of the world. He stated that it was fairly well established that T. semipenetrans was peculiar to citrus roots. Later Baines and Thorne (1) reported this organism from olive, Olea domestica, and subsequently Nesbitt (4) reported persimmon (Diospyros lotus) as a new host. Goodey and Franklin (3), citing the work of Seinhorst and Sauer, catalogued grape, Vitis vinifera, while Raski et al. (5) added lilac, Syringa vulgaris, to the host list.

Females, males and larvae of a nematode agreeing morphologically with Tylenchulus semipenetrans were recovered from the roots of climbing hempweed, Mikania batatifolia DC., in Florida. Two different collections were made from Dade county about 10 miles northwest of Homestead at a spot from which no records of cultivation are known. Climbing hempweed is a twining vinelike composite common in hammocks, low pinelands, the Everglades of south peninsular Florida, and the Florida keys. Mature females were dissected from the roots on two separate occasions while males and larvae were collected in substantial numbers by root incubation and by the Baermann funnel technique.

Inoculation experiments both to verify the host and to test the organism on citrus are planned.

A recent survey by the United States Department of Agriculture, Plant Pest Control in cooperation with the State Plant Board of Florida showed that several citrus groves in Florida appear to be in a poor physiological condition due to Tylenchulus semipenetrans. Out of 30,918 root samples collected 2787 contained Tylenchulus semipenetrans, representing 749 different properties (6). Species of Pratylenchus, Rotylenchus, Helicotylenchus, Hoplolaimus, Xiphinema, Criconemoides, Hemicycliophora, and Tylenchorhynchus which appear to be native to Florida were also found on citrus. Because of the discovery that T. semipenetrans is apparently native and also because of the number of other plant-parasitic nematodes on new planting sites it is advisable that Florida growers fumigate before planting so as to give the new plants a good start.

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STATE PLANT BOARD OF FLORIDA, GAINESVILLE

SOME INITIAL FINDINGS ON NEMATODES ON PEACHES IN NEW JERSEY[1]

H. Allen Thomas[2]

A preliminary survey of nematodes found on peaches in New Jersey included samples from 19 orchards, many of them old or declining. Several samples in each orchard were combined to provide one representative sample. The following list shows the nematode genera found and the number of orchards in which they occurred: Pratylenchus 11, Xiphinema 11, Aphelenchoides 5, Ditylenchus 4, Longidorus 2, Tylenchorhynchus 2, Criconemoides 2, Aphelenchus 2, Tylenchus 1, and Hoplolaimus 1. In practically all locations, the relative abundance of these genera corresponded roughly to the frequency of occurrence. One additional orchard was surveyed especially thoroughly, and the relationship between nematodes on these peaches and on the major crops and plants on the farm, notably corn and privet, is being studied. Most of the trees in this orchard were 15 to 20 years old and not doing well. Here the most prevalent genera were Criconemoides, Pratylenchus, Hoplolaimus, Aphelenchus, Longidorus, Tylenchus, Xiphinema, Ditylenchus, and Aphelenchoides, in descending order of prevalence. Traces of Meloidogyne and Tylenchorhynchus were also encountered.

A colony of Criconemoides was established on the roots of peach seedlings in the greenhouse, the original individuals being taken from the peach orchard mentioned above. The nematode was identified as C. xenoplax (Raski) by A. L. Taylor, United States Department of Agriculture, Beltsville, Maryland. Specimens are being used in observations and inoculations to determine the life history of this nematode on peaches. Present efforts are being directed toward defining feeding habits and the nature of the injury produced. C. xenoplax appears to feed by prolonged, uninterrupted penetration of the roots by its long stylet. As much as two-thirds of the stylet may sometimes be seen penetrating the host. No observable lesions have been produced so far. Limited egg deposition has been noted and eggs photographed. No males have been located.

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A NEW TECHNIQUE FOR PRELIMINARY SCREENING OF NEMATOCIDES

A. L. Taylor, Julius Feldmesser and W. A. Feder[1]

OBJECTIVES OF PRELIMINARY SCREENING

The principal objective in preliminary screening of chemicals for possible use as nematocides is elimination of those not toxic to nematodes and therefore obviously of no value for that purpose. Other tests are then used to select the best of the remaining compounds. Since chemicals may be toxic in the solid, liquid, or vapor phases, conditions in the preliminary tests should be such that the chemical can come into contact with the nematodes in any or all of these phases. It is also desirable that preliminary tests be designed to use very small amounts of chemicals and that the test be reproducible within reasonable limits so that a comparison of the toxicity of the test chemical and that of a standard chemical can be made.

SCREENING TECHNIQUES REPORTED PREVIOUSLY

Preliminary tests of chemicals reported in the literature have mostly been of the type called a "water screen" by McBeth and Bergeson (1). In such a test, nematodes are immersed in a water preparation of the chemical, either a solution or an emulsion. The nematodes are left immersed in the preparation in sealed hollow-ground slides for 24 hours. A similar test using a Bureau of Plant Industry Syracuse watchglass as the container was reported by Tarjan and Cheo (4). McBeth and Bergeson noted that: "The water screen technique is not infallible and should not be depended upon altogether as certain compounds highly effective by injection, especially volatile materials, have failed to show a high degree of toxicity by this method of testing."

Tarjan (3) conducted tests with several species of nematodes, both plant-parasitic and free-living, and several different chemicals, and found that Panagrellus redivivus (Linn., 1767) Goodey, 1945, a free-living nematode, was about as susceptible to the action of the chemicals as were root-knot nematodes, Meloidogyne incognita (Kofoid & White, 1919) Chitwood, 1949, which are parasites of numerous species of plants, and an undescribed Ditylenchus species, which feeds on mushroom spawn. Several other species were even more susceptible to the chemicals used. Previous experience with soil nematocides has indicated that there are differences in the degree of control that can be obtained with different combinations of nematode species and chemicals in the field. However, no chemical effective against one species of nematode has been shown to be totally ineffective against another species. Tarjan (3) concluded that Panagrellus is suitable for use in nematocide screening and is particularly convenient because of its size (up to about 1.4 mm long) and the ease with which it can be cultured.

THE NEW SCREENING METHOD

The present investigation was undertaken in the hope of devising a preliminary screening technique superior to the "water screen". The methods described were selected as the best of those tested in a series of experiments which will not be reported in detail.

Culture of Nematodes for Use in Tests

The nematodes used as test organisms are mixed populations of Rhabditis sp. and Panagrellus sp. These can be grown in oatmeal, as recommended by Tarjan (3), on sliced raw potato, on washed quartz sand, or on sterilized cellulose sponge drenched with potato broth. The last two methods are preferred, since they yield large numbers of nematodes that can be freed easily from associated organic matter. This is important since organic matter from the cultures was shown to interfere with some of the early tests, probably because of its adsorption or absorption of the chemical. It was also found that nematode populations serving as controls often failed to survive in closed vials containing large amounts of organic matter, presumably because of unfavorable conditions for respiration. As shown by Santmyer (2), Panagrellus requires considerable oxygen.

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Table 1. Nematodes on screw-top vials killed by direct contact with D-D mixture, Thimet and MOPA in 48 hours.

Chemical name and replication no.	Nematodes killed (percent) by indicated concentration of chemical (ppm).									
	125	100	80	75	50	40	25	20	10	None
D-D:										
1	100	100		100	95		37			2
2	100	100		100	99		92			7
3	100	100		100	96		63			1
4		100	100			45		15	6	0
5		100	100			89		18	26	9
6		100	100			82		22	14	12
7		100	100			61		21	10	21
8		100	100			33		15	6	12
9		100	100			93		30	16	3
10		100	100			100		97	45	*
11		100	100			73		27	24	26
12		100	100			16		26	12	17
13		100	100			74		27	4	2
14		100	100			88		70	73	25
15		100	100			95		38	24	5
16		100	100			96		36	30	10
17		100	100			89		88	81	7
18		100	100			94		75	71	14
Average	100	100	100	100	97	75	64	33	26	10
Thimet:										
1		100	100			100		84	88	9
2		100	100			95		92	72	16
3		100	100			97		76	82	15
4		100	100			99		82	86	13
5		100	98			91		86	75	10
6		100	100			90		76	68	10
7		100	100			96		59	11	10
Average		100	100			95		79	69	12
MOPA:										
1		3	6			6		14	11	9

* - Discarded in error.

Cellulose sponge pieces about 6 cm long by 2.5 cm wide by 1 cm thick, in petri dishes, are soaked in potato broth made by boiling 100 grams of fresh white potatoes in 1000 ml of water until they have disintegrated. The petri dishes containing the soaked sponges are then sterilized. Each sponge piece is inoculated with approximately 100 nematodes from a previous culture. No attempt is made to free the nematodes from associated organisms, and no special precautions are taken to prevent contamination of the cultures by bacteria or fungi. The nematodes possibly feed on organisms associated with fermentation of the medium as well as on the medium itself. Cultures are maintained at room temperatures, which vary from 15° to 25° C. Cultures contain large numbers of nematodes after about 2 weeks.

Nematodes for use in the tests are obtained by washing them from the sponges into a beaker. The contents of the beaker are then poured through a U. S. standard 60-mesh sieve

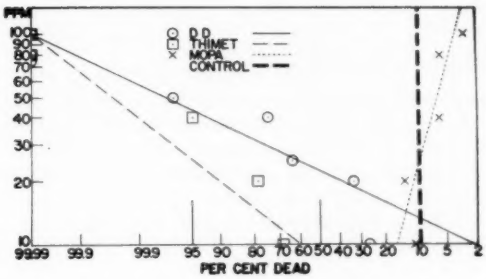

FIGURE 1. Plotted dosage-response curves secured in the in vitro vial screening technique for D-D mixture, Thimet and MOPA.

nested in a U. S. standard 200-mesh sieve. After gentle rinsing with tap water, the nematodes caught on the 200-mesh sieve are substantially free of organic matter and can be washed off into a beaker. By settling and decanting, they are concentrated until there are about 100 in 1 ml of water.

Preparation of Test Chemicals

Test chemicals are made up in standard preparations containing 1000 ppm of the technical material by volume. These may be solutions in water, water and acetone mixtures, or emulsions made by the use of various wetting agents or suspensions. Care should be exercised in the selection of solvents, wetting agents, emulsifiers, etc., used in formulation, since many of these materials are nematocidal. Minimal amounts of acetone and wetting agents such as AR-55 and AR-60 (Velsicol products) used under the conditions of the test have no nematocidal action. (Tarjan (3) found that Rohm and Haas' Triton X-45, X-100, and X-188 were nematocidal to some species and should be avoided.)

Testing Procedure

Test containers are glass vials of 5.1-ml average capacity with plastic screw caps. The testing procedure begins by adding 1 or 2 ml of the nematode suspension to each vial. Then the vials are about two-thirds filled with thoroughly washed dry quartz sand. Amounts of stock preparations of the test chemicals used are calculated so that the technical material under test occupies 10, 20, 40, 80 or 100 millionths of the volume of the vials. Thus, never more than 0.51 ml of the 1000 ppm stock preparation is added to a vial. The material is delivered into the vials from a standard model microburette calibrated in twentieths of a milliliter. The vials are shaken so that sand, nematodes, and chemical are mixed thoroughly. The vials are then filled with dry sand and the tops are screwed into place. Thus, the sand in the prepared vials is damp, not wet. A control vial with nematodes and sand but no chemical, or with the solvent, wetting agent, or other material used in the formulation, is prepared for each five vials set up with the test chemical. Test vials are stored for 48 hours at 20° C.

At the end of the test period, the contents of each vial are washed into a 100-ml beaker filled by directing a brisk stream of water to roil the sand into it. The sand is allowed to settle for a few seconds; then the supernatant fluid containing the nematodes is poured onto a coarse-porosity Büchner funnel attached to a vacuum line. This process is repeated, and the nematodes are washed from the funnel to a marked counting dish.

The nematodes are examined under a binocular dissecting microscope at magnifications of 10X to 60X. The first judgment of the effect of the test chemical can be made by observing motility. Actively moving nematodes obviously have not been killed by the chemical; and if a large percentage of the nematodes from a test vial are active, the treatment can be judged ineffective without further examination. If all or most of the nematodes are motionless, a decision must be made as to whether they are dead or paralyzed. Usually, this decision can be made if the dish containing the nematodes is set aside at room temperature for 2 or 3 days. At the end of this period, nematodes paralyzed by lack of oxygen or exposure to a test chemical will have revived and have become motile. Dead nematodes, on the other hand, will have started to disintegrate and the contents of the bodies will be darker than those of living nematodes, especially at their anterior end. With several replications of each treatment, each

with 100 to 200 nematodes, the desired information as to effect of the chemical can be obtained without making decisions as to whether each nematode is dead.

Evaluation of Test Results

Effective chemicals at the higher rates will kill all nematodes in all replications, as shown in the first four columns of Table 1. At rates less than the minimum lethal concentration, results will be erratic, as shown by the next five columns. A peculiarity of Panagrellus is that it is ovoviviparous, unlike most free-living and plant parasitic nematodes, which are oviparous. Thus, the females often contain living active larvae. This stage is the most difficult to kill by chemical or other means and, at rates equal to LD_{95} or less, will often be active even when all other stages have been killed.

Examples of Test Results

Reproducibility of results with this technique was tested with D-D mixture and Thimet, an organic thiophosphate insecticide. Results shown in Table 1 indicate that the minimum lethal concentration of D-D mixture is between 50 and 75 ppm. At 40 ppm or lower, results were highly erratic in individual replications, but averages of a number of replications show killing more or less in proportion to the application rate. Plotted on logarithmic-probability paper, these results lie near a straight line (Figure 1).

Thimet is evidently effective between 40 and 100 ppm, but there was an unexplained survival of a single nematode in one replication at 100 ppm.

The last line in Table 1 shows results obtained with MOPA (alphamethoxyphenylacetic acid). This compound is of interest because it is readily translocated in plants, but evidently it has no nematocidal action.

DISCUSSION

The screening technique described has the desirable features mentioned in the first paragraph of this paper. Conditions are such that the chemicals can demonstrate toxicity in solid, liquid, or vapor phases. As little as 100 mg of a chemical is sufficient for several replications. Tests are reproducible within reasonable limits and comparisons with standard chemicals can be made.

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AN IMPROVED TEST FOR NEMATODES IN THE SOIL

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According to a recent article in Agricultural and Food Chemistry (1), "Nematodes destroy an estimated 10% of the nation's crop annually", so it is to be expected that considerable effort would be spent in developing tests for detecting, separating, and concentrating nematodes in soil samples.

Several tests involving diverse kinds of funnels and filtration devices have been described in recent times. The most recent, devised by Miller (2), is inexpensive and simple. The "Plastic Tube" method to be described herein, like Miller's, utilizes facial tissue as the filtering medium, but is superior in some respects as shown by the following:

	Miller's Method	"Plastic Tube"
1. Size of sample of soil tested.	Limited to 5 ounce paper cup	Capacity of funnel is 16 ounce
2. Concentration of nematodes	In 15 ml of water	In 5 ml of water or less
3. Storage of nematode sample	5 ounce paper cup, and water lost by evaporation	Constricted 1/2 inch plastic tube, and no loss by evaporation
4. Cost of materials per test	About 4 cents	About 2 cents

As with all tests for nematodes the principle is the same. Nematodes penetrate the tissue holding the soil and sink down in the water in the plastic tube.

Assembly and lay-out of the parts for this method are shown in Figure 1.

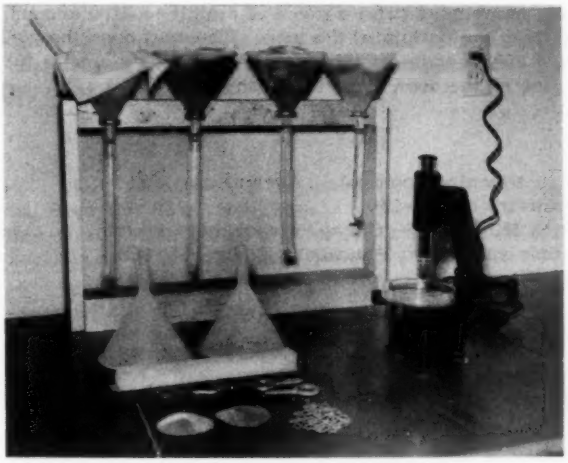

FIGURE 1. Assembly of equipment for nematode test.

[1] The complete kit, with 2 funnels, 50 plastic tubes, 2 aluminum screens, rod, screws, wire and rubber bands, may be obtained from Stoller Research Co., P. O. Box 1071, Santa Cruz, Calif.

The novel feature of this method is the use of plastic tubes, made of polyethylene, 0.002 inch thick. The tubes are 9 inches long, and 1/2 inch in diameter, or 1 inch wide when flat, and are sealed at one end. The thin plastic sheeting prevents the escape of moisture but allows exchange of the respiratory gases. Accordingly, the nematodes remain alive in the water at the end of the tube. In contrast, nematodes die or become inactive when rubber tubing is used for a similar purpose. Other advantages in the use of plastic as compared with rubber tubing are that sediment can be observed, the end of the tubing can be opened completely after constricting, constrictions can be made at several places along the plastic tube for daily observations, and the constricted end of the tube with the contained nematodes can be very conveniently stored for later observation.

Procedure for Making Test

1. Fasten a plastic tube to the spout of funnel by twisting a rubber band 3 or 4 times (plastic tube may be bent while twisting rubber band).
2. Screw the screw-type holder into a wood stand or otherwise, and insert the funnel with tube attached.
3. Fill plastic tube with water and see that there is no leak. Insert aluminum wire screen into funnel, and fill funnel with water just to surface of screen. Then take a "double" sheet of facial tissue (Scotties, Kleenex, etc.) and push the tissue down until it touches the wire screen. (Water should not touch the tissue.) Insert aluminum rod between the tissue and the side of the funnel, down to the screen.
4. Fill soil to about top of funnel. The tissue hanging over side of funnel can be turned to cover the soil; add a few drops of water to hold tissue down.
5. Push the tissue and soil away from the side of the funnel with the rod, and then pour water in the space created by pushing with the rod. Add water slowly as the soil absorbs it, and continue until free water comes to the top of funnel. In this way, no soil particles fall into plastic tube, so the water in the tube remains clear.
6. Incubate at room temperature or 70° F for 24 to 48 hours, or longer, to allow cysts to hatch.
7. To observe for nematodes twist a piece of stainless wire around the tube 1/2 to 1 inch from the bottom of the tube. Then cut open the constricted end of tube and empty contents into a glass dish. The plastic tube can be reused by fastening the open end with a band.

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STOLLER RESEARCH CO., P. O. BOX 1071, SANTA CRUZ, CALIFORNIA

BASIDIAL STAGE OF PELLICULARIA FILAMENTOSA ON BEANS

Douglas W. Burke[1]

The perfect stage of Pellicularia filamentosa (Pat.) Rogers on bean plants, Phaseolus vulgaris L., has been reported occasionally in humid regions (2). However, it apparently has not been reported previously on this host in the usually dry climate of the Rocky Mountain region.

Figure 1 shows the pellicle-like development of the fungus near the base of a bean stem. This is typical of that observed on many plants in fields near Greeley, Colorado, and in an experimental bean plot at Cheyenne, Wyoming, in September 1956.

FIGURE 1. Pellicularia filamentosa on the stem of a bean plant.

The mycelium, basidia, and spores were similar in size and morphology to those described for the fungus on potato and other crop plants (1). Mature basidia and spores were not abundant.

The unusual abundance of the perfect stage of the fungus in this region no doubt was associated with the occurrence of a period of cloudy moist weather during the first week of August, after bean foliage was heavy.

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SYSTOX-LEAD ARSENATE, ANTIBIOTICS, AND SULFUR IN THE CONTROL OF POWDERY MILDEW ON ROSES

R. S. Kirby[1]

Six plots, each containing ten rose bushes, were sprayed weekly with different materials for control of powdery mildew (Sphaerotheca) of roses, from June 15 to September 30, 1956. On all sections, excepting the sulfur-ferbam, captan at 2 pounds per 100 gallons was applied weekly but on the day before the mildew test materials. All sprays were applied with a 3-gallon compressed air sprayer.

Treatment Schedules

Section 1 - Dry wettable sulfur 4 pounds, plus ferbam 2 pounds per 100 gallons was used weekly throughout the season (June 15 through September 30).

Section 2 - Griseofulvin 100 ppm plus Acetone and Tween was used weekly throughout the season.

Section 3 - Anisomycin 100 ppm was used weekly throughout the season.

Section 4 - Anisomycin 100 ppm was used weekly in all sprays until August 11th. Starting August 25 through September 30th, Systox 12 ounces per 100 gallons was applied weekly.

Table 1. Percentage of rose leaves infected by powdery mildew.

Date examined	Section 1 Sulfur-Ferbam	Section 2 Griseofulvin	Section 3 Anisomycin	Section 4 Anisomycin	Section 5 Griseofulvin	Section 6 Systox-lead arsenate
August 16	0	0	1	0	0	0
August 22	0	0	1	1	1	0
August 27	2	6	2	29	19	0
				Starting August 25		
				Section 4 Systox	Section 5 Lead arsenate	
September 2	5	8	10	29	28	1
September 8	12	10	15	40	26	0
September 15	8	6	7	42	26	0
September 21	4	7	13	27	27	0
September 30	10	14	16	40	21	0
October 7	8	13	12	26	14	0
October 14	7	9	12	20	13	0
October 21	8	13	10	9	6	0
October 28	5	8	10	9	6	0
Average September-October	7.4	9.8	11.7	27	18.8	.11

[1] Plant Pathology Extension, Pennsylvania State University, University Park, Pennsylvania.

Section 5 - Griseofulvin 100 ppm plus Acetone and Tween was used weekly until August 11th. Starting August 25th through September 30th lead arsenate 2 pounds per 100 gallons was applied weekly.

Section 6 - Throughout the entire season, June 15th through September 30th, Systox 12 ounces plus lead arsenate 2 pounds per 100 gallons was applied in weekly sprays.

DISCUSSION

The Systox-lead arsenate combination (Section 6) gave outstanding control of powdery mildew throughout the season. Only one infected leaf was found in this section.

The control from sulfur-ferbam (Section 1) was greatly inferior to that from Systox-lead arsenate. This is shown from the average of mildew for September and October which was 7.4 percent for sulfur and 0.11 percent for the Systox-lead arsenate.

Griseofulvin and Anisomycin, with an average of 9.8 percent and 11.7 percent of the leaves infected, respectively, appear to be slightly less efficient than sulfur.

The application of lead arsenate or Systox from August 25 to September 30 appeared to check powdery mildew. However, the experimental setup does not permit a close comparison with the other materials, since the August 18 spray was omitted and Systox or lead arsenate alone were first applied on August 25th.

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A FUSARIUM ROT OF GERANIUMS AND ITS CONTROL

E. M. Stoddard

Three isolates of Fusarium sp. have been obtained from a black rot of geranium cuttings, all of which have been shown to be pathogenic by inoculation of healthy cuttings with pure cultures of the several isolates.

The diseased cuttings show a soft, coal black rot which differs from the brownish-black hard bacterial rot of geraniums caused by Xanthomonas pelargonii. It would appear that these two rots have been placed in the one category of "black leg" by geranium growers. The usual symptom of Fusarium black rot is a rapid rotting of the stem below the level of the rooting medium before roots can be produced. The line of demarcation between diseased and healthy tissue is sharply defined and apparently the infection extends only a short distance above the discolored area, as the infected cuttings when cut off above the discolored area will root readily without further stem rot. This characteristic would suggest that the organisms enter the cut end, rot the submerged part of the stem and do not become systemic. Another symptom is a rotting of the roots (with no black stem) after the rooted cuttings are potted. In this case infection takes place through roots broken in the operation of potting. Both types of infection can be produced by inoculation at appropriate times with the Fusarium isolates.

That each of the three isolates of Fusarium is pathogenic is evidenced by recovery of the organisms from cuttings inoculated with pure cultures and by reproduction of typical disease symptoms by inoculations with the recovered organisms.

Not infrequently Fusarium rot is found associated with the bacterial rot on rooted cuttings and on plants after the final potting. In the course of these studies it has been found that oxyquinoline sulfate as a soil drench gives reasonably good control of Fusarium rot and that soil drenches of streptomycin sulfate control bacterial rot. A combination of the two materials should control both diseases. Subsequent experiments have proved this to be correct; moreover, the combination of oxyquinoline sulfate and streptomycin sulfate is more effective in the control of either disease than when used alone -- an effect analogous to use of copper with streptomycin.

For these materials to be of value to the grower one should be able to cure the diseased plants and restore them to a healthy salable condition. To this end a random lot of diseased plants and rooted cuttings were collected in March 1956 from growers' stocks for experimentation. These plants were infected either with Fusarium or bacterial rot or both. All the plants were left in the soil and pots in which they were growing, and the cuttings were potted in sand and later transferred to unsterilized soil. The plants and cuttings were given five treatments at weekly intervals of a combination of 250 ppm of oxyquinoline sulfate, and 200 ppm of streptomycin sulfate, applied as a soil drench at the rate of 100 ml per 4-inch pot. Approximately 80 percent of the 40 treated plants and cuttings recovered and are in normal condition to date, while only 10 percent of the check plants are living at the present time. The treated plants received two additional applications in March 1957 to find if the materials had any effect on flowering. No effect on flowering was observed. Two lots of 25 cuttings have been made from the treated plants since February 1957. These cuttings rooted 100 percent with no evidence of stem or root rot.

Recently a large scale test of the treatment described has been made on a commercial crop of 2000 plants. All the plants were showing some degree of bacterial and/or Fusarium rot on roots and stems. The severity of disease ranged from light root infection to complete destruction of the roots. The plants received a soil drench of oxyquinoline sulfate (1.6 ounces 50 gallons) and streptomycin sulfate (200 ppm). After three weekly treatments a high percentage of the plants produced new healthy roots and showed a marked improvement in growth and appearance. Results in this test will confirm those of the original experiment.

The investigation has shown that Fusarium sp. is an active pathogen causing a "black leg" of geranium cuttings which differs in symptom expression from bacterial rot but may be mistaken for it.

Control of both the Fusarium and the bacterial black leg was obtained by using soil drenches containing a mixture of 250 ppm oxyquinoline sulfate and 200 ppm streptomycin.

The control obtained by the use of the mixture of oxyquinoline sulfate and streptomycin sulfate is due to chemotherapy and not to prophylaxis or soil sterilization.

THE CONNECTICUT AGRICULTURAL EXPERIMENT STATION, NEW HAVEN, CONNECTICUT

CHINESE EVERGREEN PLANTS REJECTED BECAUSE OF GLEBAL MASSES OF SPHAEROBOLUS STELLATUS ON FOLIAGE

Wray Birchfield, Jean L. Smith, Al Martinez, and Enid P. Matherly

Summary

The saprophytic fungus, Sphaerobolus stellatus, was observed in relation to a leaf condition of Chinese Evergreen (Aglaonema simplex) resulting from glebal deposits on the leaf surfaces of the plants. Although it was not detrimental to the plants, this condition prevented their immediate sale. Development of the fungus and the appearance of the glebal masses on the plants are discussed and illustrated.

A State Plant Board of Florida Plant Inspector, Mr. Howard M. van Pelt, at Apopka, reported a leaf condition resulting from fungal glebal deposits on the leaf surfaces of Chinese Evergreen, Aglaonema simplex L. The plants were growing in a mulch of pine and hardwood shavings which served as a substratum for a fungus. When the plants were about 2 feet in height, glebal masses of the fungus were observed deposited on both leaf surfaces of the leaf blades. About 50 to 100 masses were observed on the upper surface of single leaves, and from 6 to 20 on the lower surface. The average Chinese Evergreen has about 12 to 14 leaves. This resulted in an unsightly appearance (Fig. 1) and an unacceptable condition of the plants, a delay in marketing, and an additional cost for a "clean-up" program. Plantings of nearly an acre were involved.

The organism was identified by G. F. Weber as a saprophytic fungus, Sphaerobolus stellatus Tode ex Pers. Fischer (4), in 1884, made the first comprehensive study of its development. Buller (2) gave a report on the "shooting mechanism" of this fungus. Walker (5) made a study of the general subject.

Authorities are not in complete agreement as to the order to which this fungus belongs. It has been included by many mycologists with Nidulariales. Bessey (1) placed it in the order Sphaerobolales. Wolf (6) would prefer the Sclerodermatales, which agrees with the classification preferred by Fischer (4) and Walker(5).

The fruiting body has at least three distinct layers (Fig. 2): an outer or first peridium, an inner or second peridium, and the glebal mass. The tension arising from the osmotic swelling under specific conditions of moisture and temperature results in the violent eversion of the whole glebal mass, which may be discharged for surprisingly great distances. Walker (5) and Buller (2) showed that this glebal mass may be ejected to a distance of over four meters. As the spore fruit matures, the outer layer of the peridium splits into five lobes, in some respects resembling a minute, partially opened Geaster.

The fungus was obtained in pure culture in the laboratory by surface sterilization of the glebal masses in bichloride of mercury (1:1000 for 5 minutes) and plating on potato dextrose agar. The ejected gleba germinated, with numerous hyphae from all sides. It appeared as a white or cottony mycelial mass of very slow growth. No fruiting bodies developed on the agar. The glebal masses are spherical, as the generic name implies, about 1 to 1 1/2 mm in diameter, red in the early stage, and covered with a sticky deposit. They are filled with basidiospores. Basidia are difficult to observe. The glebal masses become dark brown or black wrinkled bodies when dried, resembling brown scale insects (Fig. 1). Prior to their discharge, a clear, watery substance accumulates around the glebal mass in the cup formed by the outer peridium. When the peridial cup is well filled, a rupture takes place and the inner (second) peridium becomes inverted over the first (outer) one, resembling a glistening opaque droplet which is dome-shaped. The glebal mass itself has no part in its discharge nor does light affect the direction of discharge, according to the studies of Walker (5). Discharge appeared to take place in the early evening and morning.

FIGURE 1. Glebal deposits of Sphaerobolus stellatus on Chinese Evergreen. Lower portion shows a close-up of these deposits.

FIGURE 2. Stages in the sporophore development of Sphaerobolus stellatus: a) immature sporophore; b) just before ejection of gleba; c) after ejection of gleba.

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STATE PLANT BOARD OF FLORIDA, GAINESVILLE, FLORIDA

NEW HOST RECORDS FOR ELSINOE LEPAGEI BITANC. & JENKINS[1]

Mortimer Cohen[2] and Albert S. Muller[3]

The first discovery of a scab on any species of the family Sapotaceae was made by H. S. Lepage at Santos, São Paulo, Brazil, in 1938. The causal fungus was described as Elsinoë lepagei Bitanc. & Jenkins on Achras sapota L.[4].

In November 1955 Mr. L. J. Daigle, Florida State Plant Board inspector, found a number of small canistel trees (Pouteria campechiana (HBK.) Baehni) in cans in a nursery at Hialeah, Florida, with heavily scabbed leaves. This material was sent to the central laboratory at Gainesville for diagnosis. Drs. A. Bitancourt and Anna E. Jenkins subsequently determined the causal agent to be E. lepagei, thus establishing that a second member of Sapotaceae is host for a scab producing fungus. Cultures of the fungus and herbarium material were then sent to the U. S. National Fungus Collections at Beltsville, Maryland, and specimens were added to the Florida Agricultural Experiment Station Herbarium (F 44863).

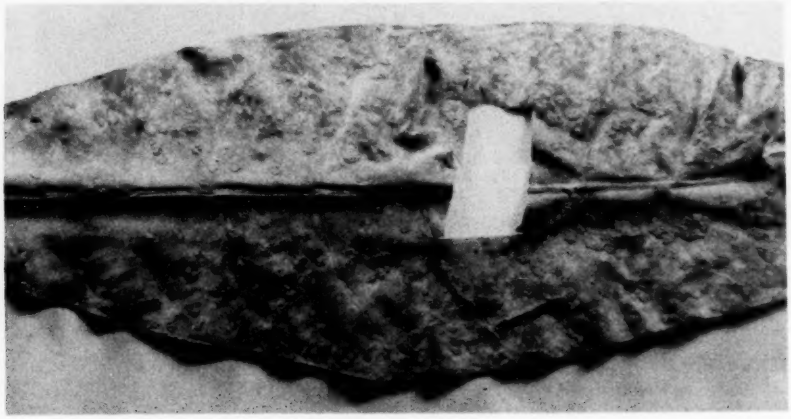

FIGURE 1. Elsinoë lepagei on Canistel

On November 16, 1956, a second observation of scab on sapodilla (Achras sapota L.) was made at Santiago de las Vegas, Cuba. Nearly all of several hundred potted nursery plants were severely scabbed. Dr. Jenkins identified the pathogen as E. lepagei and material was incorporated in the above mentioned herbaria.

Scab was found at Homestead, Florida on young grafted sapodilla trees growing in cans. These trees, found on March 6, 1957, were, at least in part, of the "Black" variety. A limited examination of bearing canistel and sapodilla trees in the Miami and Homestead, Florida areas in March 1957 did not reveal any additional instances of scab.

Scab has been found on canistel and sapodilla on either the upper or lower surfaces of the leaves, producing small, raised spots usually 0.5 to 3.0 mm in diameter. These spots are often gray at the center. Figure 1 is a photograph of a canistel leaf which has been severely attacked by scab disease.

FLORIDA AGRICULTURAL EXPERIMENT STATIONS, AND UNIVERSITY OF FLORIDA

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[2] Assistant Plant Pathologist, Florida Agricultural Experiment Stations, Citrus Station, Indian River Field Laboratory, Ft. Pierce, Florida.

[3] Professor of Plant Pathology, University of Florida, Gainesville, Florida.

[4] Bitancourt, A. A. and A. E. Jenkins. 1941. Treze novas especies de Elsinoë do Brasil. Arq. Inst. Biol. 12: 1-20. Est. 13.

OBSERVATIONS ON WITCHES'-BROOM OF HACKBERRY

Robert Snetsinger and E. B. Himelick[1]

Witches'-broom is a disease that, by causing abnormal growth of twigs and branches, is responsible for disfigurement of common hackberry, Celtis occidentalis L. This disease contributes largely to the undesirability of hackberry as a shade tree. In 1888 Kellerman and Swingle (2) associated a powdery mildew fungus and a new species of eriophyid mite with the disease. The powdery mildew was originally described as follows: "Sphaerotheca phytoptophila, Kell. & Swingle. --Mycelium very sparse: perithecia globular, dark brown or black, obscurely reticulate, 60-75 micr. in diameter; appendages few, more or less evanescent, dark brown, irregular but usually about 6 micr. in diameter and mostly longer than the diameter of perithecia, often septate. Asci large, hyaline, broadly oval, containing 8 spores, which are hyaline, oval, regular in size, 15 x 24-18 micr. Conidial stage: mycelium more abundant, conidiophores hyaline, erect total height (including conidia) 150-220 micr., by 9-13 micr. in diameter; conidia oval hyaline, continuous, granular within, 15 x 21-29, mostly 15 x 27 micr."

In the Kansas Agricultural Experiment Station Report of 1888, Kellerman and Swingle (3) actually described the eriophyid mite which they had associated with witches'-broom of hackberry, but failed to assign the mite a scientific name. Also they discussed the symptoms of the disease, the morphology of the powdery mildew fungus, and possible remedies. Halsted (1) discussed the morphology of the powdery mildew fungus. Salmon (4) reviewed the previous work, discussed the relationship between the mite and the fungus, and also described the morphology of the fungus.

Hackberry trees infested with witches'-broom bear brooms or knots composed of numerous abortive twigs (Fig. 1). The brooms, readily visible at a considerable distance, often are used by laymen as a character for identifying hackberry trees. Each broom consists of masses of twigs which bear many buds (Fig. 2, A). The twigs tend to die back after a year's growth. The buds present on the brooms are generally larger and have more open bud scales than do healthy buds (Fig. 2, B).

Throughout 1956 a study was made of an eriophyid mite (Fig. 2, C) and a powdery mildew fungus (Fig. 3) which were associated with witches'-broom of hackberry at Urbana and other Illinois locations. The morphology of the cleistothecial stage of this fungus corresponded with the description of Sphaerotheca phytoptophila given by Kellerman and Swingle in 1888. The morphology of this eriophyid also fitted the description of their unnamed mite. Specimens of the eriophyid mite from Urbana and other Illinois locations were sent to H. H. Keifer, Systematic Entomologist, Bureau of Entomology, California Department of Agriculture, for determination. The eriophyid mite apparently is a new species belonging to the genus Aceria and will be described by Keifer in the near future. The cleistothecia of the powdery mildew fungus and all stages of Aceria sp. were seen throughout the year on some hackberry trees having witches'-broom. Since the powdery mildew fungus is an obligate parasite, no attempt was made to isolate this fungus from the host tissue. Isolation attempts were made to determine if any other fungi were present, but the results were inconsistent and inconclusive.

The eriophyid mite was often extremely abundant under bud scales and inside the bud tissue; sometimes 2,000 or more mites of this species were counted per bud. Considerable damage is caused to the developing leaf tissue in the buds by the feeding of these mites. The female mites start to lay eggs in May and new generations develop throughout the spring, summer, and fall.

During the spring and summer of 1956, 44 common hackberry trees in 11 counties in Illinois and 27 Mississippi hackberry trees, C. laevigata Willd., in 3 counties in southern Illinois were studied. Thirty-nine of the 44 common hackberry trees had varying degrees of infestation of witches'-broom. Of these 39 trees, 37 had brooms with their buds infested with Aceria sp. On common hackberry trees free from witches'-broom no mites of this species were found. Fifteen of the 37 trees had buds infested with the fruiting structures of the powdery mildew fungus, S. phytoptophila. The cleistothecia of the fungus were never observed unless Aceria sp. was also present. However, the absence of fungus fruiting structures does not necessarily mean that the fungus was not present. Witches'-broom was not observed on any of the 27 Mississippi hackberry trees examined.

[1] Research Assistant in Economic Entomology and Assistant Plant Pathologist, respectively, Illinois Natural History Survey, Urbana.

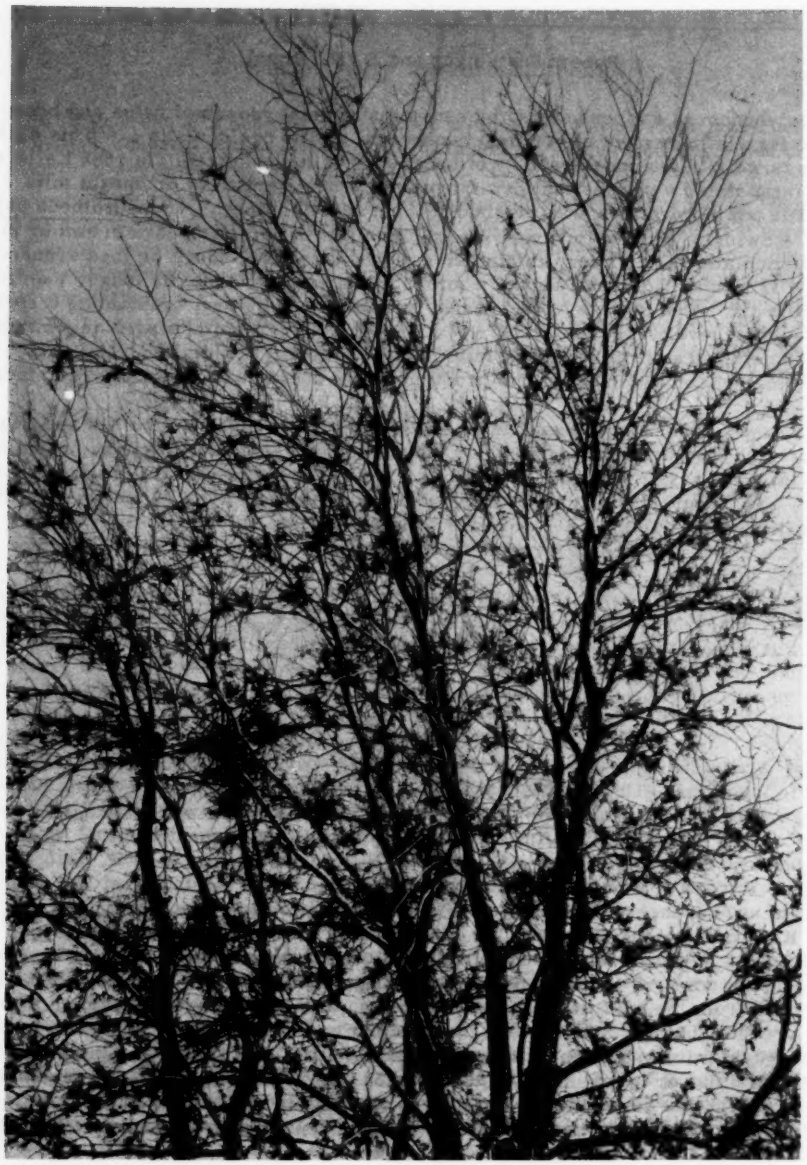

FIGURE 1. A common hackberry tree showing a heavy infestation of witches'-broom.

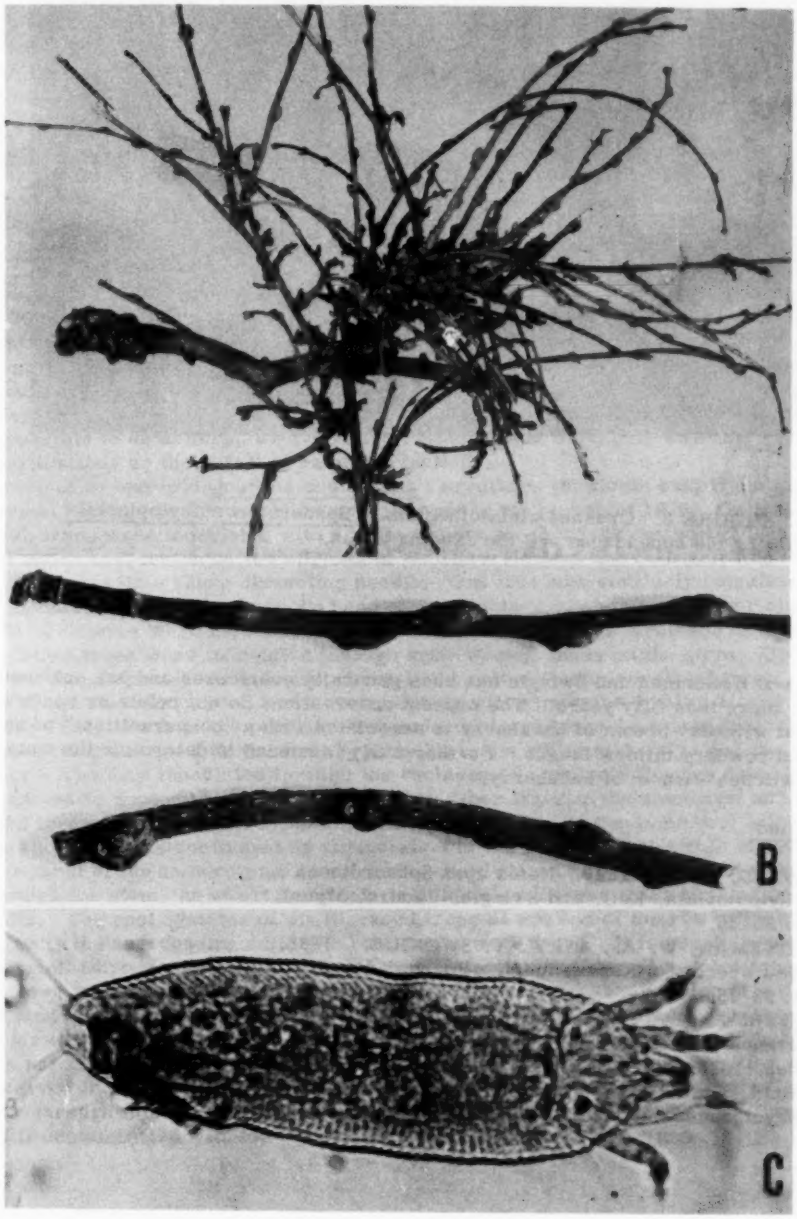

FIGURE 2. A -- A single witches'-broom or a "broom". B -- Upper twig shows normal healthy hackberry buds with tight bud scales. Lower twig shows the open type hackberry buds from trees infested with witches'-broom. C -- Aceria sp. found abundant under and inside bud scales and inside the bud tissue of most hackberry trees infested with witches'-broom.

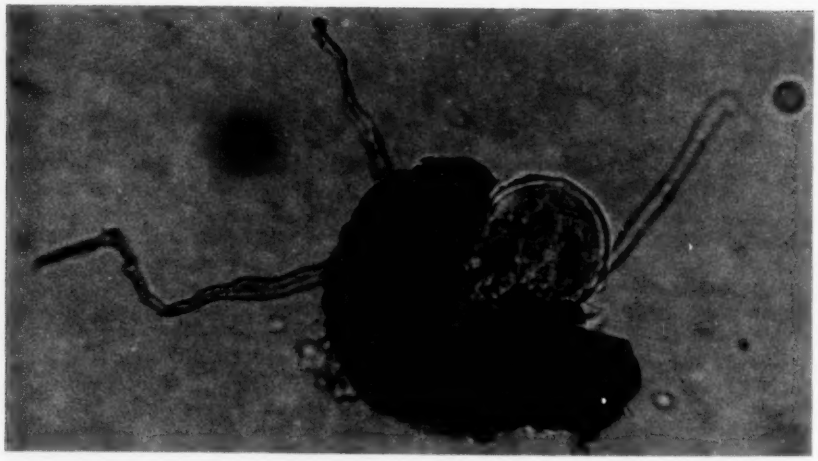

FIGURE 3. Crushed cleistothecium of Sphaerotheca phytoptophila showing the appendages and the exuding ascus.

The work of Kellerman and Swingle has been generally overlooked and has not been substantiated for more than fifty years. The present observations do not refute or confirm their contention that witches'-broom of hackberry is associated with a "co-parasitism" of an eriophyid mite and powdery mildew fungus. Further study is needed to determine the causal agent or agents of witches'-broom of hackberry.

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ILLINOIS NATURAL HISTORY SURVEY, URBANA, ILLINOIS

OAK FLOWERS MAY SERVE AS INFECTION COURTS FOR THE OAK WILT DISEASE

T. W. Bretz and T. W. Jones[1]

Oak wilt infections have resulted from the introduction of the fungus, Ceratocystis fagacearum (Bretz) Hunt, into wounds in roots, stems, twigs, leaves, and axillary buds of oak trees. Infection through inoculation of flowers has not been reported.

In the spring of 1953, attempts were made to inoculate large, forest-grown oak trees by introducing spore suspensions of the fungus into wounds in flowers. Eighteen scarlet (Quercus coccinea Muenchh.), 10 black (Q. velutina Lam.), 4 northern red (Q. rubra L.), 3 white (Q. alba L.) and 2 blackjack (Q. marilandica Muenchh.) oaks, ranging from 5 to 16 inches d.b.h., were included in the study. From 5 to 50 flowers were inoculated per tree; the average was 36 per tree. Except for two trees on which the rachises of staminate flowers were inoculated, the inoculations were made through wounds in the stigmas of pistillate flowers. Slightly more than 8 percent of the inoculated trees developed wilt. Although no other oaks in the immediate vicinity of the inoculated trees became infected, the results of the study were not entirely conclusive. Wilt occurred in the general area where the work was done, so it was not possible to determine, with certainty, whether the infections were the result of the flower inoculations or the result of natural infections.

A group of 8-year-old Quercus acutissima Carruthers (Sawtooth oak) trees growing in an experimental nursery in Central Missouri flowered in the spring of 1956. On May 8, 1956, 11 of these trees were inoculated with a white mutant strain of the oak wilt fungus by placing a drop of spore suspension on the stigmas of pistillate flowers and then pricking the stigmas through the drop with a sharp dissecting needle. One tree was similarly inoculated, except that the inoculations were made at the base of the rachises of staminate flower clusters. Fifteen to 20 flowers were inoculated on each tree. To check the virulence of the spore suspension, three trees were inoculated through knife wounds made on the stem. Uninoculated trees of the same species were interspersed in the row with the inoculated trees.

Two of the stem inoculated trees showed early wilt symptoms on May 21 and the third on May 26, 1956. Four of the pistillate-flower-inoculated trees developed typical oak wilt symptoms during the season, the first by June 27, a second in mid-July, and the other two in September. The tree inoculated through the rachises of staminate flowers developed typical wilt symptoms by September. In addition, a few other trees in the same row and close to the inoculated trees developed wilt symptoms during the course of the summer. Infection in all symptomatic trees was confirmed by re-isolation of the white mutant strain of the fungus.

Since most of the uninoculated trees that became diseased were adjacent to the trees inoculated through the stem, an effort was made to determine if these trees became infected through root grafts. The root systems of six diseased trees at one end of the row were excavated by washing away the surrounding soil. No root grafts were found. It is possible that grafts between small fibrous roots were broken before detection and that some root transmission did occur. However, two of the pistillate-flower-inoculated trees that developed wilt symptoms were located at the far end of the row from the stem-inoculated trees, a distance too great to account for their infection by root-graft transmission. The white mutant strain of the oak wilt fungus is not known to occur naturally in Missouri and no overland spread of the disease has been observed in the nursery. The results of the study thus indicate that oak wilt infections can occur through injured flowers. This suggests the possibility that certain flower-feeding insects, if contaminated with the oak wilt fungus, could serve as vectors.

MISSOURI AGRICULTURAL EXPERIMENT STATION IN COOPERATION WITH THE CENTRAL STATES FOREST EXPERIMENT STATION, FOREST SERVICE, UNITED STATES DEPARTMENT OF AGRICULTURE

[1]Respectively, Professor of Forestry, Department of Forestry, University of Missouri, Columbia, Missouri, and Pathologist, Central States Forest Experiment Station, Forest Service, United States Department of Agriculture, Columbia, Missouri. Contribution from the Central States Forest Experiment Station, Forest Service, United States Department of Agriculture, and Missouri Agricultural Experiment Station, Journal Series No. 1744. Approved by Director.

OAK WILT FUNGUS NOT FOUND IN ACORNS FROM DISEASED TREE

T. W. Bretz and W. D. Buchanan[1]

The oak wilt fungus, Ceratocystis fagacearum (Bretz) Hunt, has been isolated from the roots, stems, twigs and leaves of infected trees. A number of investigators have attempted to recover the organism from acorns of diseased trees, but no successful cases have been reported. Since most oak wilt infected trees die before an acorn crop is matured, the previous attempts by the senior author were with immature acorns. In 1956, however, mature or nearly mature acorns were obtained from a wilt infected black oak (Quercus velutina Lam.) that had been artificially inoculated by the junior author on August 1, 1956.

The tree was in advanced stages of wilt by September 13 when it was felled. Twigs bearing acorns were collected at random on September 15, and attempted isolations were made on September 17, from each twig and all of the acorns borne thereon. The twig tissues were fresh and green. The seed coats of the acorns were dry, but the embryos were full and firm. A total of 124 acorns was used in the study. Only the embryo tissues and the adhering membranous inner seed coat were plated on the isolation medium. The oak wilt fungus was recovered from approximately half of the twig samples, but it was not recovered from any of the acorns, even those that had developed on twigs in which the fungus was shown to be present. The acorn tissues were practically devoid of microflora.

Of interest was the activity shown by the acorn embryos. Black oak acorns normally remain dormant and germinate only after a period of stratification at low temperatures. This dormancy is largely because of conditions in the embryo[2]. The embryos of the acorns from the diseased tree, however, showed immediate activity in the culture dishes. Some "scar" tissue developed at points of injury to the cotyledons. There was also a rapid development and elongation of the plumule and the primary root. Secondary roots developed and there was some slight growth of the primary leaves. Some chlorophyll developed in the plumule and primary leaf tissues. To check if the activity shown by the cultured embryos was a response to injury, acorns from a disease-free black oak were similarly handled and plated in culture dishes. These embryos remained dormant and "scar" tissue developed on the injured cotyledons. This suggests that possibly some metabolite had developed in the oak wilt infected tree that accounted for the germination of the embryos and the formation of "scar" tissue on the cotyledons, or that the embryo tissues had not yet reached a stage of physiological maturation and dormancy.

The failure to recover the oak wilt fungus from the acorns further substantiates the results of previous work. Although the trials to date have been limited and cannot be considered conclusive, they do indicate that acorns that develop on diseased trees do not commonly harbor the oak wilt pathogen. Thus they probably are not important in perpetuating the disease by giving rise to diseased seedlings, nor are they likely to constitute a source of inoculum for certain insects, such as acorn weevils (Curculonidae species) which are known to infest acorns.

MISSOURI AGRICULTURAL EXPERIMENT STATION IN COOPERATION WITH THE CENTRAL STATES FOREST EXPERIMENT STATION, FOREST SERVICE, UNITED STATES DEPARTMENT OF AGRICULTURE

[1] Respectively, Professor of Forestry, Department of Forestry, University of Missouri, Columbia, Missouri, and Entomologist, Central States Forest Experiment Station, Forest Service, United States Department of Agriculture, Columbia, Missouri. Contribution from the Central States Forest Experiment Station, Forest Service, United States Department of Agriculture, and Missouri Agricultural Experiment Station, Journal Series No. 1745. Approved by Director.

[2] Woody-Plant Seed Manual. U. S. Dept. Agr., Forest Service, Misc. Pub. No. 654. 1948.

NEW RECORDS OF PLANT DISEASES

CAMELLIA PETAL BLIGHT FOUND IN TEXAS

By A. G. Plakidas[1]

On April 6, 1957, a specimen consisting of four Camellia japonica diseased blossoms (varieties not named) were received from Mrs. Nell Rowan, Longview, Texas, for diagnosis. The blossoms showed typical symptoms of petal blight, and the causal fungus, Sclerotinia camelliae Hara, was readily isolated from them.

Texas, thus, is added to the growing list of southern states (Louisiana, Georgia, South Carolina, North Carolina, Virginia) into which this fearful disease has been introduced. I use the term "introduced" purposely in order to stress the fact that in every case in which the disease was found in the South it was traced to introduction in that particular locality of potted or balled camellia plants from California. It is evident, from the context of Mrs. Rowan's letter, that in this particular case also the blight was introduced on potted camellia plants from California. She writes:

"I have been raising camellias for some twenty years without any difficulty until I got what I think is petal blight. Three years ago I bought some beautiful camellias in cans from California. That year I had a trace of spots.....last year it got worse, and this year it has been awful."

There is a strong probability that the blight will be found in other localities in Texas if a search is made for it. Texas has no quarantine against the importation of potted camellias, and it is known that potted or balled camellia plants have been imported from California on many occasions.

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A NEW RUST FOR THE WEST INDIES

By Alice J. Watson[1]

In the course of a survey in Puerto Rico for local plant diseases conducted by Albert L. Brown of the Plant Quarantine Division, Agricultural Research Service, two collections were made of a rust fungus on leaves of St. Augustine grass (Stenotaphrum secundatum). This fungus, present only in the uredial stage on these specimens, has been determined as Puccinia stenotaphri Cummins and the identification confirmed by Dr. Cummins. Darluca filum (Biv.) Cast. was also present on the rust pustules. This rust was first described in the uredial stage by Sydow in 1909 from Central Africa and the telial stage was first noted and described by Cummins on material from Mauritius. The Puerto Rican specimens appear to constitute the first report of the occurrence of this rust species in the Western Hemisphere. The host is abundant and widespread throughout the West Indies and the Southern United States and additional localities for the rust may be expected.

PLANT QUARANTINE DIVISION, AGRICULTURAL RESEARCH SERVICE, UNITED STATES DEPARTMENT OF AGRICULTURE, BELTSVILLE, MARYLAND
[1]Associate Mycologist, Plant Quarantine Division.

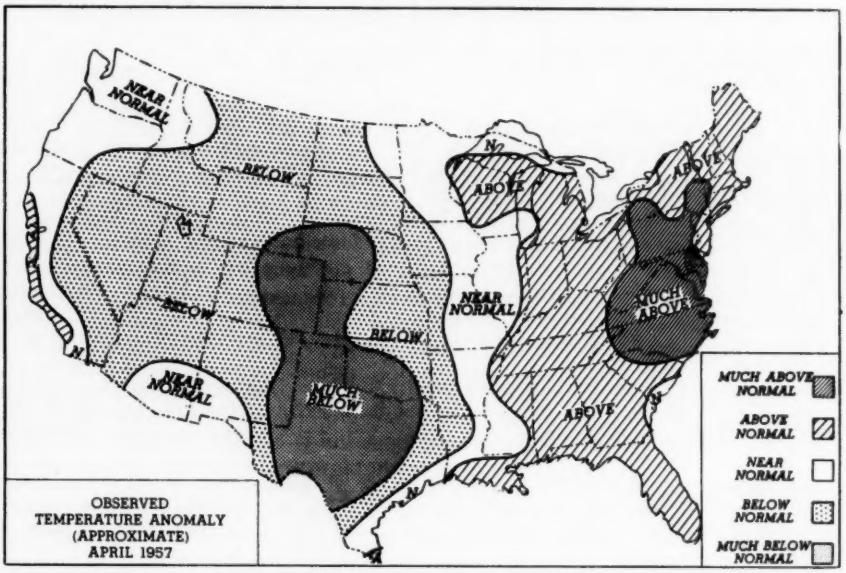
SOME CORRECTIONS

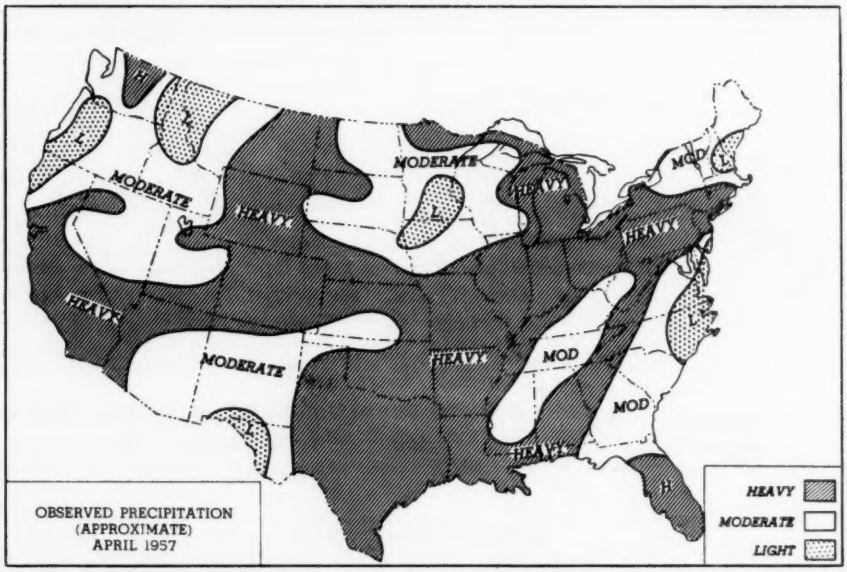
REPORTER, Vol. 41, No. 4, April 1957, article by H. H. McKinney, W. R. Paden, and Benjamin Koehler, "Studies on chemical control and overseasoning of, and natural inoculation with, the soil-borne viruses of wheat and oats." On page 264, line 7, counting from the bottom of the page, delete the word due.

REPORTER, Vol. 41, No. 4, April 1957, article by V. H. Young, Jr., A. M. Somerville, Jr., and J. L. Carnes, "Nematode Control of Potted Ornamentals with V-C 13 Nemacide." On page 275, figure 4 should read ".....A3, infested potting soil treated with V-C 13 two weeks before transplanting. Plants photographed 13 months after treatment."

IN THIS ISSUE (Continued).

New records of plant diseases, page 547: Camellia petal blight found in Texas, by A. G. PLADIKAS; A new rust from the West Indies, by ALICE J. WATSON. Some corrections, page 547. April weather, page 548. Manuscripts for the Reporter, page 549.





The terms used in the accompanying maps, which define the ranges of temperature and precipitation, are numerical class limits. These are based on a statistical analysis of past records through which is determined the normal frequency of occurrence of temperatures and precipitation at various times of the year for different locations. For temperature the classes above, below, and near normal are so defined that they each normally occur one-fourth of the time; much above and much below normal, one-eighth of the time Precipitation is depicted in terms of light, moderate, and heavy, each class normally occurring one-third of the time and thereby having equal probability of occurrence. These maps graphically represent only the general trends and give the country's weather picture at a glance. For quantitative studies, where monthly mean temperatures and actual precipitation records are needed for a given time and place, other publications of the Weather Bureau should be consulted. P. R. M.

MANUSCRIPTS FOR THE PLANT DISEASE REPORTER

(See also PDR 35: 121-122; 36: 214, 494)

(1) GENERAL: The Reporter page measures 9 inches long with the heading or 8 3/4 inches for the text part, by 6 inches wide. The copy is typed on a larger page, 11 1/4 inches of text or 12 inches overall in length by 8 inches in width, and reduced 25 percent in the photographic process of reproduction. Illustrations or tables larger in either dimension will take a correspondingly greater reduction. Only one size of type is available for text, footnotes, or tables.

(2) MANUSCRIPTS should be the original ribbon copy, not carbons, clearly typed and double-spaced throughout, including tables, footnotes, and bibliographies. (Note -- only one copy is needed.) Footnotes should be typed at the bottom of the page.

(3) ABSTRACTS are requested for all except very short articles. (PDR 38: 316).

(4) CAUSES OF DISEASES should be named. For bacteria, fungi, nematodes, etc., give the Latin name of the organism; for viruses either or both the accepted common name of the virus or a Latin name if you prefer it and there is one; for non-parasitic diseases state the causal factor if it is known. If the cause of a disease has not been determined say so. (PDR 36: 494).

(5) LITERATURE REFERENCES should be given in alphabetical order and numbered for citation in the text. We follow the AIBS suggestion of placing the year of publication after the author's name. Please check your titles carefully since we cannot do it for you. Be sure that text citations and bibliography agree; that foreign-language references are correct; that number or month is cited for periodicals that are not paged consecutively throughout the volume.

(6) NAMES OF FUNGICIDES should be given according to the suggestions that will be published in Phytopathology before long, and later repeated in the Reporter. In the meantime see PDR 35: 122.

(7) ILLUSTRATIONS should be sent to us unmounted. To prevent mistakes, write figure numbers on the back, and mark the top of each print when necessary. A sketch can show a preferred arrangement but please keep in mind page size, shape, and standard reduction (see above under General), and remember that figure titles and legends are part of the page. Lettering should be clear and large enough to be legible after reducing. Drawings, maps, and graphs can be photographs or originals, but should be finished and ready for reproduction, not just sketches.

(8) TABLES should be carefully thought out with particular attention to the Reporter's limitations in reproduction. Make titles and headings definite and self-explanatory. Designate footnotes in tables with superscript lower-case letters. Be sure that text discussion agrees with the data in the table. Do not abbreviate names of crop varieties.

(9) REPRINTS cannot be supplied since there is no way in which we can be reimbursed. However,

(10) The MULTILITH PLATES from which reprints can be made will be sent if requested at the time the article is submitted. The press size of these plates used for the Reporter is designated as small -- maximum image 9 1/2 by 13 inches, maximum paper size 9 3/4 by 14 inches -- for Model 1250. Most of the Experiment Stations have this type of multilith machine.

(11) We are the official correspondents in the United States of the PLANT PROTECTION BULLETIN published monthly by the United Nations Food and Agriculture Organization. We would appreciate it if plant pathologists would send us suitable material from time to time for inclusion in our monthly summary reports. Please mark it clearly so that we will know it is intended for the Bulletin. (See PDR 36: 492; 37: 637) -- P. R. M.

XU